JUL 10 1923

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THE

# MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—10TH YEAR. SYDNEY: SATURDAY, APRIL 21, 1923. No. 16.

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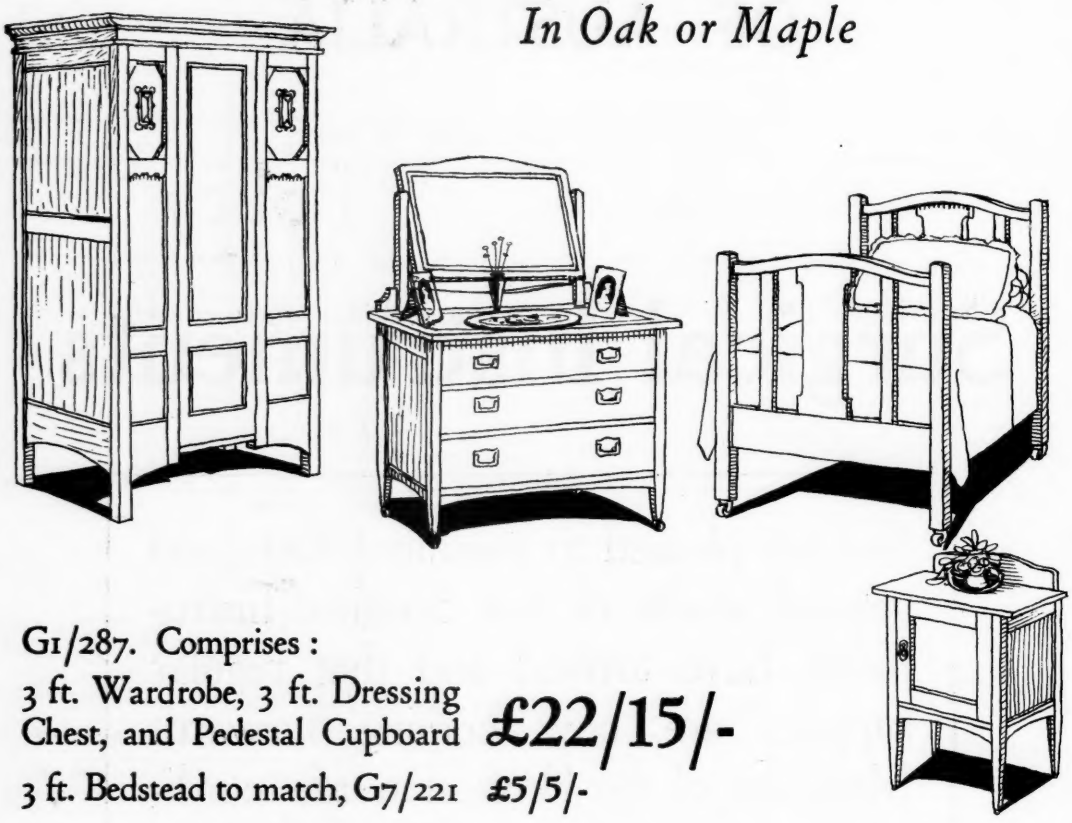

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# THE MEDICAL JOURNAL OF AUSTRALIA

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## A FURTHER SERIES OF CASES CLOSELY RESEMBLING TYPHUS FEVER.[1]

BY FRANK S. HONE, B.A., M.B., B.S.,

*Chief Quarantine Officer for South Australia; Honorary Physician, Adelaide Hospital; Lecturer in Preventive Medicine and in Clinical Medicine at Adelaide University.*

AT the monthly meeting of this Branch in July, 1921, a discussion took place on a series of cases closely resembling typhus fever which had been noticed in and around Adelaide since early in 1918. Of some thirty cases which were known to have occurred, sixteen were then reported in detail, with *post mortem* notes of three fatal cases that had been observed.[2] In THE MEDICAL JOURNAL OF AUSTRALIA (January 19, 1922) I reported a further case which I had seen in conjunction with Dr. Wall.

As I now have a record of twenty additional cases which I know to have been under observation in the last twelve months, I have thought it might be of interest to bring this matter again before members and to review our present knowledge of the problem.

From Table I. it will be seen that these cases have been observed by some eight or ten practitioners. Partly through their kindness and partly through being fortunate enough to have four patients in my ward, I have personally seen the majority of the rashes described. The full details of the Adelaide Hospital cases will be found in the "Adelaide Hospital Archives," 1923. I append details of two private cases as described by their respective medical attendants. Other cases than those mentioned here may probably have been attended by other practitioners. The fact that within twelve months of attention being directed to the matter twenty cases have been reported, whereas in the four preceding years detailed notes of only sixteen cases were obtained, suggests either that the disease is slowly spreading or that cases have been passing unrecognized.

### ÆTIOLOGY.

#### Locality.

It was noted in the original paper that all the cases observed in the first year had occurred in the Port Adelaide district, but that the more recent cases had arisen at Lockleys, Norwood and in Adelaide. Of the twenty patients now under discussion, five lived within the boundaries of the city of Adelaide, two at East Adelaide, one each at Nailsworth, Medindie, North Adelaide, Unley, Keswick, one at Bowden, six at Hindmarsh (five in one group).

Curiously enough, none has been recorded to my knowledge in the Port Adelaide district since April,

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 22, 1923.

[2] THE MEDICAL JOURNAL OF AUSTRALIA, January 7, 1922.

TABLE I..

| No. | Initials. | Sex. | Age. | Residence. | Occupation. | Medical Attendant. | Onset. | Appearance of Rash. | Temperature Normal. | Weil-Felix Test. | | Complication. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | Date. | Agglutination | |
| 1. | Mr. C. | Male | 45 years | Maylands | —— | Dr. E. A. Brummitt | Jan. — | —— | —— | Feb. 3 | C 1: 120 | —— |
| 2. | G.A.[1] | Male | 56 years | Bowden | Deep Drainage Labourer | Dr. F. S. Hone | Feb. 16 | Feb. 22 | Mar. 3 | Feb. 24<br>Feb. 27<br>Mar. 7<br>Mar. 14<br>Mar. 30 | None<br>None<br>C 1: 40<br>C 1: 40<br>C 1: 320 | Pleurisy |
| 3. | W.McC. | Male | 40 years | Flinders Street, Adelaide | Labourer | Dr. Glen H. Burnell | Mar. 5 | Mar. 11 | Mar. 15 | Mar. 13<br>Mar. 23 | C 1: 1,280<br>C 1: 640 | —— |
| 4. | B.R.[1] | Female | 57 years | Franklin Street, Adelaide | Housework | Dr. C. T. C. De Crespigny | Mar. 18 | Mar. 24 | Apr. 2 | Mar. 28<br>Apr. 13 | None<br>C 1: 2,560 | —— |
| 5. | K. | Male | —— | North Adelaide | Labourer | Dr. E. A. Brummitt | Apr. 5 | Apr. 11 | —— | Apr. 11 | C 1: 80 | —— |
| 6. | C.M.[1 2] | Male | 14 years | 69, Port Road, Hindmarsh | Baker | Dr. J. H. Evans and Dr. F. S. Hone | Mar. 24 | Mar. 29 | Apr. 7 | Apr. 6<br>Apr. 10<br>Apr. 12<br>Apr. 24 | C 1: 40<br>C 1: 320<br>C 1: 320<br>None | —— |
| 7. | E.M.[2] | Female | —— | 69, Port Road, Hindmarsh | Housework | Dr. C. F. Pitcher | Apr. 2 | —— | —— | Not Done | —— | —— |
| 8. | A.M.[2] | Male | 65 years | 69, Port Road, Hindmarsh | Saloon Keeper | Dr. J. H. Evans | Apr. 13 | Apr. 19 | —— | Not Done | —— | —— |
| 9. | A.K.[1 2] | Female | —— | Next door to 69, Port Road, Hindmarsh | Housework | Dr. F. S. Hone | May 21 | —— | June 2 | June 12 | C 1: 160 | —— |
| 10. | D.M.[2] | Female | 10 years | 69, Port Road, Hindmarsh | Housework | Dr. C. F. Pitcher | June 9 | —— | —— | Not Done | —— | —— |
| 11. | H.P. | Male | 20 years | Unley | Baker | Dr. F. S. Scott | May 19 | May 25 | —— | May 27 | C 1: 160 | —— |
| 12. | C.W. | Male | 30 years | Nailsworth | Linesman | Dr. E. A. Brummitt | June 7 | June 13 | June 15 | Not Done | —— | —— |
| 13. | Mrs. E.[1] | Female | 27 years | Waymouth Street, Adelaide | Housework | Dr. F. S. Hone | June 3 | June 7-8 | June 17 | June 12<br>June 27 | C 1: 80<br>C 1: 640 | —— |
| 14. | Mrs. P.[1] | Female | 52 years | East Adelaide | Housework | Dr. F. S. Hone | ? | ? | Aug. 2 | Aug. 3<br>Aug. 8<br>Aug. 15 | C 1: 3,200<br>C 1: 5,000<br>C 1: 1,280 | Pneumonia |
| 15. | S.L.J. | Male | 29 years | Walkerville | Grain Merchant | Dr. P. S. Messent | Aug. 3 | Sept. 4 | Sept. 14 | Sept. 12 | C 1: 160 | —— |
| 16. | G.W.[1] | Female | 22 years | ? | Housework | Dr. D. R. W. Cowan | Sept. 18 | Sept. 21 | Oct. 3 | Sept. 29<br>Oct. 6<br>Oct. 13 | C 1: 5,120<br>C 1: 10,240<br>C 1: 320 | —— |
| 17. | W.L.[1] | Male | 54 years | Sturt Street, Adelaide | ? | Dr. J. Johnson | Oct. 1 | ? | Oct. 19 | Oct. 25 | C 1: 2,560 | Pneumonia on October 21 |
| 18. | McC. | Male | —— | Keswick | —— | Dr. W. T. Hayward, Dr. W. H. Collins, Dr. John P. O'Brien | —— | —— | —— | Nov. 13<br>Nov. 25 | None<br>C 1: 320 | —— |
| 19. | A.K. | Male | 38 years | Hindley Street, Adelaide | Boot Repairer | Dr. A. E. Shepherd | Dec. 12 | —— | —— | Jan. 4 | C 1: 160 | —— |
| 20. | B.McC. | Male | 15 years | Hindmarsh | Engineer | Dr. J. H. Evans | Jan. 29 | —— | —— | Jan. 10 | C 1: 640 | —— |

C = Complete agglutination.

[1] These patients were treated at the Adelaide Hospital. [2] Hindmarsh group of patients.

1919. I have again during the past year made frequent inquiries of country practitioners in this State, but can learn of no cases in the country resembling these we have seen. I have also on different occasions inquired of physicians in Melbourne and Sydney, who tell me that neither in private practice nor in hospital records have any similar cases yet been seen, although a careful look out has been kept. One case of typhus fever appeared in the health records of Queensland last August, but official inquiries in that State could elicit no information concerning it. With the information at present at our disposal, therefore, it would seem that this disease became prominent in Port Adelaide in 1918, but for the last two years has been more or less endemic in and around Adelaide.

### Seasonal Incidence.

The present series of cases have been distributed fairly evenly through the year, with some special prevalence last autumn.

I have tabulated these with the previous seventeen cases of which we have details, and find that the thirty-seven cases were distributed among the months as follows:

Table II.—Seasonal Incidence.

| Month. | 1918. | 1919. | 1920. | 1921. | 1922. | 1923. | Total. |
|---|---|---|---|---|---|---|---|
| January | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| February | 2 | 2 | 0 | 0 | 1 | 0 | 5 |
| March | 0 | 1 | 0 | 1 | 3 | 0 | 5 |
| April | 0 | 1 | 0 | 0 | 3 | 0 | 4 |
| May | 0 | 0 | 0 | 1 | 2 | 0 | 3 |
| June | 1 | 0 | 0 | 2 | 3 | 0 | 6 |
| July | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| August | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| Sept. | 1 | 0 | 0 | 0 | 2 | 0 | 3 |
| October | 1 | 0 | 0 | 0 | 1 | 0 | 2 |
| Nov. | 1 | 0 | 0 | 0 | 1 | 0 | 2 |
| Dec. | 1 | 0 | 0 | 1 | 1 | 0 | 3 |
| Total | 8 | 4 | 0 | 5 | 19 | 1 | 37 |

If these figures mean anything, the greatest incidence is in late summer and autumn. Typhus fever, it will be remembered, is commonest in winter, chiefly because the crowding together that naturally occurs then in the poorer quarters of cities affords better opportunity for spread by the body louse.

It will be noticed that in this small set of figures July and August come the lowest of any two months in the year. It is interesting that the seasonal incidence approaches that of typhoid fever rather than typhus. If infection is due to an insect intermediary, any seasonal variation would only depend on the life habits of that insect. Last year's experience would suggest a crop of cases in the next few months, which is one reason for directing attention to the matter at this juncture.

### Age.

The majority of the cases observed, as before, have been in adults, although one of the patients was aged fourteen and another ten years. It will be noticed that both of these were in the group of five at Hindmarsh. A possible explanation of this age incidence is that, like typhus, cases in children are mild and often missed. South African observers have emphasized this in the typhus fever that has been prevalent there of recent years and in a newly published work on the Russian epidemic of typhus fever in 1919-1920, Mitchell lays stress on the same point. Such mild missed cases, with very faint rash, may be an explanation of the continued occurrence of the disease.

### Sex.

Whereas all the previous cases recorded were in males and even one or two supposed cases in females proved on searching the records to be clearly some other disease, yet in the present series no less than seven out of the twenty patients were females. Three of these were in the group at Hindmarsh where the original infection occurred in a youth, but the others were isolated cases, where no previous case could be found in the house. An interesting point is that in all these female patients the affection was very mild; two of the three in the Hindmarsh group were nursed at home; two in the Adelaide Hospital would not have been noticed if we had not been keeping a sharp look-out and the third patient only came in with a mild bronchopneumonia that had supervened when the rash was beginning to fade. In each case the rash was scanty. This adds weight to the suspicion that a case occurring in a woman may often be missed.

## Symptomatology.

### Type of Cases.

Although no deaths have occurred in this series and these cases have on the whole been of a mild type, the same general features have been observed as in the previous series.

Although the illness has not always started suddenly, it will be noticed that in most cases the patient can point to the exact day on which he felt ill. Most of the patients have not been seen until after a few days' illness, but where records have been kept from the beginning, the temperature has mounted rapidly, reaching its maximum of from 38.3° C. to 39.4° C. in a day or two. Apart from the pyrexia and its accompanying symptoms, the most prominent symptom during the first week has been the headache.

I stated previously that I had come to regard this as a characteristic symptom of the first week; further experience has confirmed this statement. It is generally frontal and is complained of much more than the headache in typhoid fever or other febrile conditions. It generally becomes a little easier after the rash appears, but may persist for ten days. Dr. Burnell's patient on the seventh day stated: "It comes on in spasms every ten minutes"; the man I saw with Dr. Brummitt at Nailsworth was very emphatic about it. Another patient (a woman) said it was like a nerve jumping, always worse when she was trying to sleep. It nearly always needs medical treatment for relief. In the first five days physi-

[1] These patients were treated at the Adelaide Hospital. [2] Hindmarsh group of patients.

cal examination reveals nothing distinctive. Vomiting has frequently occurred in the first few days; the state of the bowels varies; epistaxis has only once been noted. The pulse is generally slow compared with the pyrexia; with a temperature of 38.3° C. in one case it was 76, in another 68. I have generally noticed it under 100 even with pyrexia of 39.4° C., but in one or two cases it was said to have been 110 to 114. This was generally later in the disease. Respiration is not specially affected. There may be slight cough, but I have not noticed those signs of bronchial catarrh which are so common in typhoid fever.

#### Condition of the Spleen.

A careful watch has been kept on the spleen; in some of the cases it has been quite definitely palpable. In the case of Mrs. P., who was admitted to hospital when the rash was beginning to fade, it was still palpable and was increased in the next day or two. In this case it was hard and firm like the spleen in malaria.

#### Toxæmia.

By the time the rash appears the patient is generally in a toxic condition, out of all proportion to his pyrexia and pulse rate. Even in the mildest case, he is generally a little delirious at night during the first week. It is noted as before how quickly this condition clears up as the time of defervescence approaches. Defervescence, as before, has occurred about the twelfth to the fourteenth days, by rapid lysis in this series rather than by crisis.

#### Rash.

The rash has appeared quite definitely on the fifth to seventh day. The first macules are scattered and are generally mistaken for typhoid spots, but by the second day of the rash they are too widely distributed for this and have the appearance of a mild measles rash, although the macules are not so closely set together. They continue to come out for two or three days. In these mild cases the petechial spots have not been so distinct; they appear later than the macules. The subcutaneous discoloration, also, has not been so apparent in some cases, although when the rash is fully developed the description of a "dirty measly rash" which was previously given, holds good. In the milder cases the rash will very easily escape notice, especially in a dark room. I have been struck by the fact that it quite often has to be pointed out in the first case seen by a particular physician, but once seen, men easily recognize it as present in subsequent patients, which they would have otherwise missed. The most striking instance of this was a house surgeon of mine, who quite missed the rash in his first patient, until it was pointed out to him. A month later (when we had only seen one case in a woman) a woman was admitted to the ward with an indefinite history of a week's illness and a very definite history for twenty-four hours, pointing to pneumonia. Examination revealed consolidation at the base of the right lung, but he noticed that the abdominal *striæ atrophicæ* were unduly pink and noted that "subcuticular mottling of the skin causes *striæ albicantes* to show up pink and obscure a faint rash which was papular over the abdomen with some macules. Some spots disappear on pressure." Upon this he had the blood examined and found that it completely agglutinated *Bacillus proteus X 19* in a dilution of 1 in 2,560.

The description of the rash in the Adelaide Hospital cases will be found in the "Hospital Archives." Dr. Burnell says in the notes of his case "on the chest, abdomen and arms are a few spots something like measles, which fade on pressure. On the back are similar macules." Two days later the notes read: "Rash almost gone, leaving a few stains which do not quite disappear on pressure." In the case of S.L.J., Dr. Messent reports that numerous rose-coloured papules were scattered over the abdomen, chest and back; the spots faded completely on pressure. They were typical typhoid spots, but very numerous. He counted one hundred on the abdomen and back. The patient had not noticed them until that morning. Two days later he wrote that the spots were numerous and in some places were confluent macules and papules; the rash was on the arms and thighs. Four days later the rash was reported to be fading.

#### *Distribution of Rash.*

It will be seen that the rash begins on the abdomen and chest and then appears on the back. It is always most pronounced here, but extends to the upper arms and thighs. Until Drs. Collins and O'Brien kindly showed me a patient under the care of Dr. W. T. Hayward at Keswick Repatriation Hospital in November, I had not seen the spots extend beyond this. In that patient, however, the rash went right down both arms and legs to the back of the hands and feet. Spots were also present on the face. I afterwards learnt that the rash in Dr. Scott's patient had the same distribution; it was thickly distributed on the face and right down the extremities to the hands and feet. The Keswick patient was in a highly toxic condition, bearing out the suggestion that the extent of the rash bears some relation to the intensity of the toxæmia.

#### Condition of the Conjunctivæ.

Particular attention has been paid to the conjunctivæ and in the majority of these patients it has been noticed to be injected. In some this is quite a distinct feature and an aid to diagnosis.

#### Blood Changes.

All the patients examined in hospital have shown a leucocytosis in the first week, generally about 9,000 to 10,000. After we had established this, I had a letter from Professor Hoffmann, of the West Indies (who had read the account of the previous discussion), in which he said that he had learnt to use this as a means to distinguish typhoid from typhus. There is no doubt that it serves to distinguish these cases we are seeing from typhoid fever, even before the rash appears. The differential white cell count has shown no alteration in the proportion of different kinds of leucocytes.

In the case of A.K., who developed broncho-pneumonia just before admission to hospital, the count revealed 16,000 white cells, probably because of the complication. This is borne out by the case of A.L., who was admitted when apparently in the third week of the disease, with a few petechial spots left on his abdomen. His temperature was normal on the day of admission. Three days later his temperature rose to 38.3° C., signs of consolidation appeared at the base of his lung and his white count ran up from 10,000 to 23,000. In the uncomplicated cases, thus, the leucocyte count averages about 10,000, sufficient to distinguish it from typhoid fever.

### Weil-Felix Reaction.

In all the Adelaide Hospital patients and in the majority of the patients seen outside, the blood has been tested for its power of agglutinating *Bacillus proteus X 19*. In the fifteen instances in which the test has been applied, such agglutination has occurred in dilutions varying from 1 in 80 to 1 in 10,000. In no case has the serum agglutinated the *Bacillus typhosus* or *Bacillus paratyphosus A* or *B*.

At the same time Dr. Bull of the Adelaide Hospital Laboratory has done a routine Weil-Felix test in all cases of typhoid fever and in many other cases of pyrexia in which the Widal test has been called for. Of seventy such cases thus investigated, no agglutination of *Bacillus proteus X 19* has occurred beyond a dilution of 1 in 40. Even these have been only occasional and when repeated have not been sustained.

This serological test is quite definite, therefore, for this series of cases and can be relied on as a diagnostic in cases of doubtful eruption. The patient whom Dr. J. H. Evans sent to my ward just after the previous discussion, had pyrexia and a rash somewhat similar to the one described while he was working among wool bales infested with "wool weevils"; this test was applied, but no agglutination was obtained. The subsequent clinical course also differentiated it.

In the case in the series also of G.P., seen by Dr. Scott and Dr. De Crespigny, the test performed on the eighth day, when the rash was fully out, yielded a reaction. His brother had been similarly ill for six days except for the rash. His serum did not agglutinate *Bacillus proteus X 19*. It was thought at the time that a rash would develope later, but no rash appeared and Dr. Scott tells me that the subsequent course was quite different.

Two precautions must be observed. (*i.*) The stage in the disease at which the agglutinins appear seems to vary considerably. The power of agglutination may be absent or only very slightly present before the rash appears. Even during the week of the rash the patient's blood may only agglutinate partially or in a dilution of 1 in 60 or 80. But if the test be tried again a week later, when defervescence has taken place, agglutination will occur in much higher dilutions. In those few cases of other disease where a doubtful agglutination occurred on one occasion, this increase never developed. This agglutination in higher dilutions as convalesence approaches is exactly the same as occurs in typhus fever. Adopting the standard laid down by Reynolds of calling an agglutination in dilution of 1 in 100 occurring after the eighth day a reaction for typhus fever, it will be seen that all the patients gave a reaction, although not always as early as the eighth day.

(*ii.*) The same technique must be carefully followed in all cases.

In the previous series of cases the tests reported had been done at the Commonwealth Serum Laboratories. A certain amount of confusion occurred in the first case of this series, because in the first test done at the Adelaide Hospital Laboratory there was a doubtful or no result (probably from being early in the disease). A test a week later in Melbourne gave a positive result; repeated a week later at Adelaide the result was not so clear. It was then found that the culture was being maintained at 54° C. in Adelaide and 37° C. in Melbourne. Subsequent investigations in other cases showed that this difference of temperature appreciably affected results. Dr. Bull describes the technique in his summary of these tests.

### Complications.

The only complications noted in this series were: (*i.*) pleurisy during convalescence in one patient and (*ii.*) pneumonia.

In one patient this occurred when the rash was fully out and did not seem to affect the course of the disease; in the other patient it came on three days after pyrexia had ceased. In both the pneumonia was quite different from that of the three patients in the first series who died of a terminal pneumonia after defervescence had set in while the toxæmia persisted.

### Treatment.

Treatment has mainly been along the general lines of pyrexial conditions. Any special dieting, as for typhoid fever, has naturally been abandoned. Rather, in view of the asthenia following some of the previous cases, an effort has been made to give as large a number of calories as possible in the dietary suitable for pyrexia. Whether as a result of this or of the mildness of the affection, convalescence has been rapid in all the patients.

As has been stated, headache has generally called for special medicinal treatment, the coal tar derivatives being found most successful.

### Diagnosis.

The disease for which these cases are still most often mistaken, is typhoid fever. It is a queer swing back, when we realize that seventy years ago all cases of typhoid fever were being labelled as typhus and considerable efforts had to be made to get them finally differentiated. Now we find ourselves lumping together under the name of typhoid, cases that clinically and serologically resemble typhus.

The diagnosis from typhoid fever ought to be made in the first week by the more sudden onset and the leucocytosis. In the second week the differential diagnosis depends on the wider distribu-

tion of a more profuse rash, the absence of agglutination to the Widal test and the reaction to the Weil-Felix test.

From measles the longer train of symptoms before the appearance of the rash and the absence of coryzal symptoms ought to make the diagnosis evident. The rash, while sometimes closely resembling measles at first, in its subsequent development appears different and should lead to a Weil-Felix test being done.

One case in this series shows the necessity of remembering that pneumonia with a history of a week's previous illness may be only a complication of an infection of this sort, whose presence would be revealed by a Weil-Felix test being made.

Occasional difficulty arises in saying whether the rash is the true rash of this disease or not. This difficulty was particularly felt in the case at Keswick. The patient was at first thought to have been continuously in hospital for over two weeks before onset; his rash was both on the face and the extremities, which I had not seen before. He had a chronic bone condition which might have set up some septic blood state; his temperature had not been very regular. The first Weil-Felix test here was undeterminate; but the second, done a week later, left no doubt as to the diagnosis.

### Discussion.

Our further experience, therefore, during the past year has confirmed the opinion as to the close resemblance both clinically and serologically of these cases with typhus fever. Does this resemblance extend to the mode of transmission of the disease?

The latest authorities re-emphasize the part played by the louse in the transmission of true typhus, as originally discovered by Nicolle. Fitzgerald, at Toronto, in the most recently published book on "Preventive Medicine," says that it is generally believed that typhus is not conveyed in any other way than by the louse, head lice and body lice both serving as vectors. Mitchell, Asheshov and Richardson, who have quite recently published a volume dealing with their experience in Russian epidemics of typhus fever, say that it is only in the presence of the louse that patients can transmit infection.

In this further series of cases of ours no evidence of infestation with body lice was ever discovered and in only one case is the presence of head lice recorded. On the other hand, the belief is strengthened that the *Bacillus proteus X 19* is only a secondary invader in typhus fever. Dr. Cilento tells me that the Weil-Felix reaction occurs also in Rocky Mountains spotted fever and many think that the Rickettsia bodies are the causal organism in that disease as well as in true typhus. Schultz says that the Rickettsia seem to constitute a new group of micro-organisms, probably more closely related to the protozoa than the bacteria.

If this is so, a group of diseases may exist, due to these organisms, resembling one another in their response to the Weil-Felix test and transmitted by the agency of insects: typhus by the louse, Rocky Mountain spotted fever by the tick and so on. J. W. D. Megaw has described a series of cases resembling typhus fever in which he puts down the transmitting agency as a species of tick (*Indian Medical Gazette*, October, 1921, pages 361 to 371).

From the preventive aspect, therefore, the important question is to ascertain, if possible, how the infection is transmitted. In cases arising at irregular intervals like these appear to do, two means suggest themselves as possible:

(*i.*) That they are transmitted, like lobar pneumonia, by direct infection through carriers.

(*ii.*) That there is an insect intermediary host.

### Epidemiology.

As has been previously stated, these cases have now occurred in men and women varying from ten years of age to sixty-five in Adelaide, practically in all its suburbs and in all seasons of the year. The patients in hospital have been nursed in general wards with no special precautions and no further cases have occurred in other patients or nurses. The same has been true of the patients nursed at home, except in the one group of cases at Hindmarsh, which will be dealt with at greater length. The occupation of most of the women has been household work, except for two of the women at Hindmarsh, who served in the same shop. Two of the men were labourers, one a baker, one a linesman on the telegraph lines, one a saloon-keeper, one a grain merchant, one a boot repairer.

The apparent connexion with insect-infested wheat has broken down. On the other hand, while it may be only a coincidence, it is interesting to note that, as compared with any twenty individuals picked at random from the community, there still seems to be an undue relation to wheat or grain. One was a grain merchant, two were in a bake-house and the group of five at Hindmarsh occupied adjacent houses in close proximity to a bakery.

In the others, negative evidence is not of much value. For, assuming the incubation period to be somewhere about fourteen days, as seems to be the case, and remembering that one does not usually see the patient till late in the first week, one must in any individual case, be able to account for all his movements on some problematical date about three weeks beforehand. The difficulty of doing this was illustrated in the Keswick patient. This man was a returned soldier who had been in Keswick Repatriation Hospital on and off for some two years. He had been admitted a clear fourteen days before the onset of this illness. He was very torpid and delirious when the real nature of his illness was at first suggested. It was then thought he had not been out on leave during his probable incubation period. It was subsequently found that he had been out on several afternoons, but, although these were apparently the only opportunities of him picking up infection (as no other cases occurred in the hospital), the most diligent inquiries by Dr. Collins and Dr. O'Brien failed to discover all his movements on those days of leave.

Recognizing this, it was felt early that the best hope lay in securing a group of cases and there lay

the interest in the five cases which occurred in Hindmarsh.

As the first case occurred in a cool drink and sweet shop, it came under the control of Dr. Borthwick, Medical Officer of Health for the Metropolitan County Board of Food and Drugs, and, as soon as he knew that more than one case occurred, his officials made full inquiries. Subjoined is this information:

*Hindmarsh Group.*

I. C.M., aged fourteen years, living at 69, Port Road, Hindmarsh, in a wine saloon with a cool drink and sweet shop adjoining it, was reported by Dr. Evans as suffering from enteric fever on March 31, 1922. He was first taken ill on March 26, 1922. He was employed at the wholesale bakery, next door, on Saturday mornings until a week before he was taken ill and then on the "Premier" ice cream cart at Ethelton. He was last at work on March 25, 1922. He had had no meals away from home. He had eaten walnut creams which were said to be "weevily" and which had been obtained from Menz's in January. The milk supply was from White & Toogood. There was no wheat or other cereals on the premises. The chaff for horses was obtained at the wholesale bakery where the patient worked. He was removed to Adelaide Hospital on March 31, 1922, and his room was closed until it was disinfected on April 1, 1922. He was discharged from hospital on April 25, 1922. There were seven other inmates in the house.

II. E.M., the mother of the previous patient, lived in the same house and nursed the patient. Her infection was not reported. She was first taken ill on April 2, 1922. She had not been away anywhere for some weeks and had had no food away from home. She had not eaten any walnut creams or other "suspect" food. She had not been in contact with any "suspect" matter. She was nursed at home by A.K. (IV.). No disinfection of the room had been carried out.

III. A.M., aged sixty-five years, living at 69, Port Road, in the same house as the two previous patients, was reported by Dr. Evans as suffering from enteric fever on April 19, 1922. He was taken ill on April 13, 1922. He was employed in the wine saloon on the premises. He had not been away anywhere, not eaten walnut creams nor any "suspect" food, nor been in contact with any "suspect" matter. He had his clothes trunk in C.M.'s room, but slept in another room. He had entered C.M.'s room once during the latter's illness to obtain a razor strop. He was nursed by A.K. for a few days and was removed to Parkwynd Hospital on April 18, 1922. His room was closed until it was disinfected on April 20, 1922. He was discharged from hospital on May 9, 1922.

IV. A.K. lived in the house adjoining 69, Port Road, which she visited frequently to assist in nursing E.M.. Her illness was not reported. She was taken ill on May 21, 1922. She had not been away anywhere. She had not eaten walnut cream or any "suspect" food. She had not been in contact with any "suspect" matter. Her milk supply was from White & Toogood. She was removed to Adelaide Hospital on May 28, 1922. No disinfection of her room was carried out. She was discharged from hospital on June 11, 1922. There were three other inmates of this house.

V. D.M., agen ten years, was a sister of C.M. and lived in the same house. She had also visited the house of A.K. Her illness was not reported. She was first taken ill on June 9, 1922. She had not been away anywhere; she had not eaten walnut cream or any "suspect" food. She had not been in contact with any "suspect" matter. She was only slightly ill and was nursed at home. No disinfection of her room was carried out.

The irregular intervals between these cases will be noted. The mother of the first patient fell ill seven days after him and two days after his removal to hospital. She was nursed at home by a neighbour. Eleven days after her first symptoms the third case occurred. This patient was removed to hospital five days later and the neighbour who had nursed him, as well as the second patient, did not show symptoms till thirty-one days after his removal and about twenty-one days after the convalescence of the second patient. Then nineteen days later the girl in the house became ill.

This makes it very difficult to imagine any direct transmission from one patient to another, but rather suggests some infective focus on the premises. The house was in a well-sewered area, as were the homes of all the other patients of this series, and there were no sanitary defects. The first boy had confessed to a great fondness for the walnut lollies, which he said were "weevily" and these were at first suspected, but afterwards ruled out of court.

In the patient whom I saw in private, I made particular inquiries, which will show the difficulty of the problem:

This man was a patient of Dr. Brummitt's. He lived in a new house which had only been built three months. It was one of a row of similar completely detached new cottages in their own plots of ground. The road was unmade. The cottages on the opposite side were less numerous. It had been wet for two or three months and there was no dust. The place was well sewered. The man, his bed and surroundings were particularly clean. His wife had been trained as a nurse and was of a very clean type. His occupation was a telegraph linesman, so that he spent most of his days a good many feet above the ground. He had been employed for some three weeks previously on the telephone lines at Cheltenham, but had not travelled in a train or tram, always riding his bicycle to and from work. He took his lunch with him, so that he never had meals in restaurants or entered shops.

Dr. Borthwick has made similar inquiries about the last two patients who have recently had the complaint. Through his courtesy I am allowed to publish the particulars gained by these inquiries, although they still fail to reveal the source of infection:

A.K., aged thirty-eight years, residing at 133, Hindley Street, was reported by Dr. Shepherd on January 3, 1923, to be suffering from "typhoid fever" (?). On January 5 the diagnosis was changed to "typhus fever" (?), which was definitely confirmed on January 8, 1923.

The patient was confined to bed on December 28, 1922, but had been complaining for a fortnight previously. He was removed to Parkwynd Private Hospital on January 1, 1923, and was reported to be convalescent on January 10, 1923. The vacated premises were disinfected on January 4, 1923, on the presumption that the illness had been typhoid fever.

The patient had not been in contact with any person known or suspected of having the disease. He had not been recently bitten by any insect or been in contact with any verminous person. He had not been out of Adelaide for months, except to attend the races at Cheltenham on December 26, 1922, and December 28, 1922. He had not visited Port Adelaide for many years and had no friends there. His occupation was boot repairing and was carried on at his Hindley Street residence and at the City Central Market.

The Hindley Street residence was an unregistered lodging house. Fifteen Maltese men had come from various parts of the State (Port Pirie and Loveday, on the River Murray) and from Broken Hill for the Christmas holidays on December 19, 1922, and stayed there for two weeks. They had all gone at the date of inspection of the house on January 3, 1923, and very little information could be obtained about them. It appears that they slept on the premises, but had their meals elsewhere.

It was stated that some of the men were wheat lumpers at Port Pirie. As far as could be ascertained, there had been no sickness of any sort amongst them and they were very cleanly in their habits. This latter statement was borne out by inspection of the rooms and the bedding. No other lodgers had been taken in for some weeks.

Thirty-five other Maltese men had been accommodated at No. 127, Hindley Street (also belonging to K) and the above remarks apply equally to them. K. had not been in contact with any of the Maltese men.

The house occupied by K. was inspected on January 3, 1923, and on subsequent occasions was found to be exceptionally clean for that class of property. There was no indication of rat, mice or insect infestation. No animals were kept on the premises. A bag of chaff was found in the Hindley Street boot repairing shop. It was the custom to obtain a bag of chaff at intervals from a chaff store in Morphett Street. This store was inspected and showed no indication of rat, mice or insect infestation.

The leather used by K. in his trade was traced to its source in two leather warehouses in the city, but in neither of these was there any indication of rat, mice or insect infestation.

The same remarks apply to a stable in Morpett Street where K. stabled his horse.

In the case of B.M., whose illness was reported by Dr. J. H. Evans as a case of Brill's disease to the Hindmarsh Local Board of Health, the Metropolitan County Board visited the house on February 7, 1923, and the following facts were ascertained:

The patient was a lad of fifteen years of age living at the corner of Coglin Street and Port Road. He had been taken ill on January 29, 1923, and was isolated at home. He had been employed at the Union Engineering Company's works at Southwark up to January 27, 1923.

The family (consisting of two adults and two children) had been to Mypolonga for ten days at Christmas time.

The patient had recently been eating a moderate quantity of fruit (chiefly peaches and bananas) and chocolates concerning which no complaints were forthcoming.

The dwelling house was clean and well ventilated and the existence of vermin (fleas, lice, bugs) could not be established, except in the case of cockroaches, which were sometimes seen in the house. Two dogs were kept about the place and they appeared healthy and clean looking. Some fowls were kept in the yard and were said to be free from tick; they were fed on house refuse and food scraps. No wheat, chaff or pollard was kept on the premises. Rats from an adjoining pughole were occasionally seen on the premises.

The sanitary inspector of the Hindmarsh Local Board of Health informed the nurse that he had inspected the patient's place of employment and found nothing to which exception could be taken.

I have narrated these inquiries at length in order show the field that has to be covered. The crucial factors in the transmission of these cases still elude us. I feel that they must be sought along two paths. We are hoping for more intensive study of the movements and so forth of patients who may come into the Hospital and have arranged for inoculation experiments. For these to be effective, we need to see the patients earlier in the disease than we have been doing. I am also hopeful that arrangements at present in vain may result in a trained epidemiologist being set free to do the necessary field work in cases recognized outside. For these inquiries to be effective, we want to know of every doubtful and mild case as early as possible and I should be very glad of the assistance of members in giving information of every such case.

*Patient Seen by Dr. Glen H. Burnell.*

The following notes have reference to Dr. Glen H. Burnell's patient:

W.McC., a labourer, aged forty years, residing at 176, Flinders Street, was seen on March 11, 1922. His illness commenced three weeks before with a "bilious attack," characterized by headache, nausea and vomiting. He had had a slight cough, but this had disappeared. He was very thirsty. For the past six days he had had no appetite and had vomited everything except soda water. He had taken no solid food. The bowels had been opened daily till March 10, 1922. The motions were not frequent, but very loose, like soup. There was no epistaxis. The headache was slightly less at the time of examination, but was still severe. There was no abdominal pain. Micturition was normal; there was no increased frequency. On March 8, 1922, he had had a shivering attack.

The patient had enjoyed good health. He had had measles in 1902. His wife stated that he had been slightly delirious at night for a few days before. He had been at home in Adelaide for a month before the onset of the illness, doing no work. He had not been working near wheat nor had he noticed weevils. He had always been a labourer.

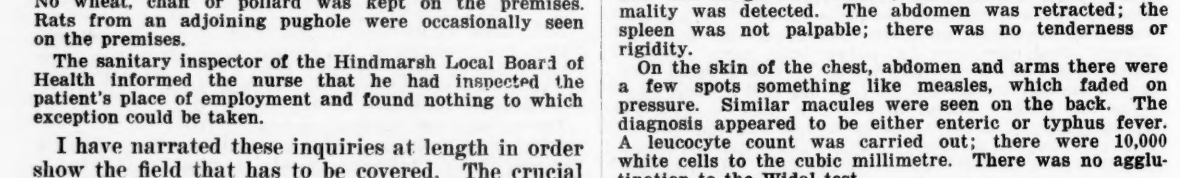
His face was slightly flushed. There was no jaundice and no coryza, but the conjunctivæ were injected. The tongue was furred. The patient had many carious teeth. The left pupil was much enlarged. This condition had been present for many years. Both pupils reacted to light.

His temperature was 38.7° C.. The rate of the pulse was 88; it was regular and of good volume and tension. The apex beat of the heart was not displaced; the sounds were muffled; no bruit was heard. The percussion note over the lungs was normal; on auscultation no abnormality was detected. The abdomen was retracted; the spleen was not palpable; there was no tenderness or rigidity.

On the skin of the chest, abdomen and arms there were a few spots something like measles, which faded on pressure. Similar macules were seen on the back. The diagnosis appeared to be either enteric or typhus fever. A leucocyte count was carried out; there were 10,000 white cells to the cubic millimetre. There was no agglutination to the Widal test.

On March 11, 1922, the patient still had headache coming on in spasms about every ten minutes. His temperature was 38.2° C., his pulse rate 84 and his respirations 29. The heart and lungs were normal.

On March 13, 1922, the temperature was 38.3° C. and the pulse rate 76. The knee jerks were brisk and equal. There was slight rigidity of the neck and a slight Kernig's sign. The rash was almost gone, leaving a few stains which did not quite disappear on pressure. The headache was still troublesome, but the patient slept better. Blood was taken for the Widal and Weil-Felix tests.



On March 14, 1922, the headache was still very bad. The patient had slept very little. His temperature was 37.8° C. and his pulse rate 76. Dr. Bull, of the Adelaide Hospital, reported that no reaction was obtained to the Widal and Weil-Felix tests. He pointed out, however, that, although there was no agglutination with *Bacillus proteus* in two hours, there was complete agglutination in 1 in 1,280 after standing over night. From the Commonwealth Serum Laboratories it was reported that a Weil-Felix reaction (1 in 320 after two hours and 1 in 640 over night) was obtained.

On March 15, 1922, the temperature was normal. The headache was a little easier. The patient had slight delirium during the night.

On March 16 the temperature was still normal. His appetite was improving and his headache was gone.

On March 23 blood was taken again. In two hours at 56° C. it yielded complete agglutination 1 in 320 and almost complete 1 in 640.

*Patient Seen by Dr. P. S. Messent,*

The notes of Dr. P. S. Messent's patient are as follows:

S.L.J., aged twenty-nine years, a grain merchant, living at Walkerville, came under treatment on September 5, 1922. Since August 30, 1922, the patient had felt excessively tired, but had been attending work. He did not care what happened. He felt drowsy, but could not sleep. He had a slight headache. He had had several shivering attacks during the previous three days. There had been no epistaxis. The bowels were regular. There was anorexia, but no abdominal pain and no vomiting.

The patient had had no previous illnesses.

His temperature was 40° C., his pulse rate 100 and his respirations 20. He was a thin man and looked exceedingly tired and drowsy. His tongue was clean. His heart and lungs were normal. There was a slight, dry cough, but no expectoration. The spleen was enlarged and hard; there was no tenderness nor distension. Numerous rose-coloured papules were scattered over the abdomen, chest and back. The spots faded completely on pressure (typical typhoid spots, but very numerous; one hundred were counted on the abdomen alone). The patient had not noticed the rash until the morning of the fifth. The specific gravity of the urine was 1,018; it contained a cloud of albumin, but no sugar nor pus. His serum did not agglutinate typhoid or paratyphoid bacilli.

On September 7, 1922, the patient was sleeping with the aid of hypnotics. There was no delirium. The spots were more numerous and in some places confluent. The rash consisted of macules and papules and was distributed over the arms and thighs. The cough was troublesome and there was some viscid, muco-purulent expectoration.

On September 11, 1922, the rash was fading. The patient still looked drowsy.

On September 12, 1922, no rash was visible and there was no staining of the skin left. The blood serum yielded a Weil-Felix reaction in a dilution of 1 in 160.

On September 14 the patient felt and looked quite well and the cough had ceased.

He stated that there were very few weevils in the wheat this year and was positive that weevils did not "bite."

**Post Scriptum.**

On March 1, 1923 (a week after reading this paper), I was asked by Dr. E. A. Brummitt to see W.B., aged thirty-five years, a tramway motorman living in East Adelaide. He had beeen first seen on February 17, 1923, on account of abdominal pain and severe headache, which had come on the day before (February 16). For a week he had been feeling tired and languid, lying down whenever possible. He had shown irregular pyrexia from February 17; on February 19 or 20 there had been jaundice, with bile and albumin in the urine. There was then much tenderness over the gall bladder. The spleen had not been palpable, nor was the abdomen distended. Scattered spots like a typhoid rash had appeared on February 25. This led to a small specimen of blood being taken for examination which failed to give a Widal reaction against *Bacillus typhosus*, but microscopically gave complete agglutination against *Bacillus proteus X 19* in dilution of 1 in 60. A larger sample of blood was then taken on February 27, which gave against *Bacillus proteus X 19* a complete agglutination in a dilution of 1 in 320, partial in 1 in 640, slight in 1 in 1,280. When I saw him on March 1 he was improving, his temperature was only 37.8° C. (100° F.), his tongue was clearing, but the headache was still bad; the toxæmia was lessening. He subsequently convalesced rapidly.

The special features of interest were:

1. Onset with jaundice, thus resembling the patient seen in May, 1920 (Case XIII., THE MEDICAL JOURNAL OF AUSTRALIA, January 7, 1922).

2. Scantiness of rash.

3. The patient lived with his wife in the same house as Mrs. P. (Case XIV. in above series), who was ill with the same disease in August last. There have been no other cases of illness during this interval in the same house, which is an isolated building two hundred yards from any other house, with a rubbish tip close by swarming with rats, which also infest the ground underneath the house.

## SEROLOGICAL EXAMINATIONS.[1]

BY LIONEL B. BULL, D.V.Sc.,
*Deputy Director, South Australian Government Laboratory of Pathology and Bacteriology, Adelaide Hospital.*

IT has been possible by the employment of a simple laboratory test to obtain certain evidence which helps in the differentiation between the disease described by Dr. Hone and other diseases presenting a somewhat similar clinical picture. The test employed is that of Weil and Felix. It consists of titrating the serum of the patient against an emulsion of *Bacillus proteus* isolated by Weil and Felix and named *"X 19."* The emulsion employed is standardized to contain from 3,000 to 4,000 million bacilli per cubic centimetre. This is made by washing the growth from an eighteen to twenty hours old agar culture with 0.9% sodium chloride solution and diluting until the desired strength is obtained. The patient's serum is diluted in saline solution. A series of small tubes is set up and one volume of saline solution is added to all except the first. One volume of diluted serum is then added to the first and second tubes and one volume is taken from the second to the third and from the third to the fourth tube and so on to the end of the series. One volume of bacillary emulsion is then added to each tube, the last tube containing no serum and acting as a control of the emulsion. The contents of the tubes are thoroughly mixed and the tubes are then placed in a water bath, kept at 37° C. for two hours. The results are read after the tubes have stood for two hours at room temperature and again after they have remained in the room overnight.

When the test was first begun, it was our practice to incubate at 54° C. for two hours, but later it was found that some sera gave higher titrations when the incubation had been at 37° C.. When the

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 22, 1923.

tubes are allowed to stand overnight before the final reading is made, a clearer definition of the result is obtained and we have never observed any tendency to spontaneous sedimentation under these conditions.

During the last fourteen months it has been our practice to titrate against *Bacillus proteus* all sera received for the purpose of the Widal test against *Bacillus typhosus* and *Bacilli paratyphosi A* and *B*. Serum from patients suffering from pneumonia has also been tested. The object of these examinations has been to determine the specificity of the test. Seventy samples of serum from patients with enteric fever, pneumonia and pyrexias of unknown origin have been tested during the year 1922. Some of these sera have been re-examined after an interval of a week or more. It has been found that occasionally such sera will produce complete agglutination in a dilution of 1 in 20 and rarely in a dilution of 1 in 40. In contrast to this finding, the sera from cases which present the clinical features of a mild typhus fever, will agglutinate the emulsion of *Bacillus proteus X 19* in very high dilution, a dilution of 1 in 10,480 being the highest yet observed to give complete agglutination. These agglutinins may appear in the blood about the eighth to the twelfth day, but may be weak. Occasionally agglutinins may not be demonstrable until late in convalescence. After a maximum has been reached, they begin to weaken and this may be manifested by incomplete sedimentation, although agglutination be present even in very high dilution. It would appear that in the majority of cases the agglutinins disappear in from four to five weeks after the commencement of the illness. The above points are indicated in the accompanying table.

The number of cases examined is small and great difficulty is experienced in getting patients to consent to having specimens of blood collected after they have left the Hospital. Under the circumstances, it has not been possible to investigate the rise and fall of agglutinins satisfactorily.

There seems to be little doubt that these agglutinins resemble the heterologous agglutinins in the typhus patient's serum. They differ from agglutinins produced by inoculating an animal with a suspension of bacilli. Wilson (1922) has shown that the agglutinins in the typhus serum are destroyed by heating to 65° C. and that emulsions of *Bacillus proteus X 19* heated to 56° C. lose their agglutinability; but according to others this agglutinability is restored by heating to 60° C. and 80° C. or 100° C.. We have found that the serum from some of the patients suffering from the typhus-like condition under review loses its agglutinins after being heated to 56° C. for thirty minutes. When a shorter period of fifteen minutes is used, some sera will completely lose their agglutinins, while in others the agglutinins are incompletely destroyed. Probably other sera will be found to lose their agglutinins only at higher temperatures. We have found that emulsions of *Bacillus proteus X 19* are no longer agglutinable after having been heated to 56° C. for one hour and that further heating to 80° C. or 100° C. does not restore the agglutinability.

If it can be shown that the serum of typhus patients does not lose its agglutinins for *Bacillus proteus X 19* when heated to 56° C. for half an hour, but only at 65° C., then such a test might be of some value in differentiating between typhus and the typhus-like disease under review. It is claimed by Wilson that agglutinins can be demonstrated with almost unerring accuracy at the end of the first week in the typhus patient's serum. This may be another point of departure between the two diseases.

The results of these agglutination tests indicate

| Patient. | Sex. | Days Ill. | Pyrexia. | Weil-Felix Reaction. |
|---|---|---|---|---|
| G.A. .. .. .. | Male | 9 | + | No agglutination |
| | | 12 | + | No agglutination |
| | | 27 | None | C 1 in 40; P 1 in 160 |
| | | 33 | None | C 1 in 320; P 1 in 640 |
| C.M. .. .. .. | Male | 12 | + | C 1 in 40; P 1 in 80 |
| | | 16 | None | C 1 in 320; P to 1 in 2,560 |
| | | 18 | None | C 1 in 320; P to 1 in 2,560 |
| | | 30 | None | No agglutination |
| A.K. .. .. .. | Female | 22 | None | C 1 in 160; P 1 in 320 |
| O.E. .. .. .. | Female | 9 | + | P up to 1 in 80 |
| | | 24 | None | 1 in 640; no change 1 in 1,280 |
| B.R. .. .. .. | Female | 11 | + | No agglutination |
| | | 16 | None | C 1 in 2,560 |
| H.P. .. .. .. | Female | 11 or more | None | C 1 in 3,200; no change in 1 in 6,400 |
| | | 16 or more | None | C 1 in 5,120; P in 10,240 |
| | | 23 or more | None | C 1 in 1,280; P in 2,560 |
| G.W. .. .. .. | Female | 10 | + | C 1 in 5,120; P 1 in 10,240 |
| | | 17 | None | C 1 in 10,240; P 1 in 40,960 |
| | | 24 | None | C 1 in 320; A.C. 1 in 2,560; P to 1 in 20,480 |
| W.L. .. .. .. | Male | 21 | None | C 1 in 10,480; P 1 in 20,960 |
| W.McC. .. .. | Male | 9 to 14 | + | C 1 in 1,280; no change 1 in 2,560 |
| | | 26 or more | None | C 1 in 640; A.C. 1 in 1,280; P 1 in 2,560 |
| K. .. .. .. | Male | 7 or 8 | + | C 1 in 80; P 1 in 320 |
| H.P. .. .. .. | Male | 8 | + | C 1 in 160; no change 1 in 320 |
| C.B.McC. .. .. | Male | — | — | No agglutination; C 1 in 320; P 1 in 640 |
| S.L.J. .. .. .. | Male | 13 | + | C 1 in 160; P 1 in 320 |

C = Complete agglutination with sedimentation. A.C. = Almost complete. P = Partial.

the value of a routine examination of all sera of patients with pyrexia where the diagnosis remains in doubt. By the means of this test we have at times been able to give the first indication to the clinician that the patient is probably suffering from a typhus-like disease. We have never found a serum giving any agglutination of *Bacillus typhosus* or *Bacillus paratyphosus* in addition to agglutination of *Bacillus proteus*. In many cases where the clinical picture is not clear-cut, a diagnosis may rest on the Weil-Felix reaction.

We have no knowledge of this test having been performed as a routine examination in any other part of Australia and, until this is done, it cannot be claimed with any degree of certitude that the disease under review is not to be found elsewhere in Australia than in this State. In performing such a routine examination it is essential to bear in mind that (*i.*) the bacillary emulsion should not be formolized or preserved in other ways, but must be made from young living cultures of the bacillus (Wilson's method of using desiccated bacilli has not been tried); (*ii.*) repeated examinations of serum from the same patient at intervals of two or more days may be necessary in order to detect the presence of agglutinins and (*iii.*) no serum should be considered as giving a positive reaction unless agglutinating in a dilution of over 1 in 40 and it should be demonstrated that the titre of agglutination rises in early convalescence. It has been found that a serum giving a high titre of agglutinins may be preserved when collected aseptically and kept in the ice-chest. It has been our practice for some time to use such a serum as a control of the agglutinability of the bacillary emulsion prepared daily.

All the tests described above have been performed by Miss E. Cleland.

**Reference.**

W. J. Wilson: "The Serological Test in Typhus: A Stable and Sensitive Diagnosticum," *The Lancet*, 1922, Volume I., pages 222 and 223.

## POSSIBILITIES IN THE TRANSMISSION OF ONCHOCERCA GIBSONI.[1]

Recorded by R. W. Cilento, M.D., B.S., D.T.M. & H., *Director, Australian Institute of Tropical Medicine, Townsville.*

The recorded observations on the transmission possibilities of *Onchocerca gibsoni* have so far failed to yield any definite result and it is thought that the facts here presented may to some slight degree help to elucidate this obscure problem.

It is recognized that there is a wide margin of error and it is not claimed that the observations now recorded are in themselves scientifically accurate, but they are of notable significance.

In the first place, at a sale of cattle at Kingaroy on November 23, 1917, Mr. Rheuben, Meat Inspector, to whom I am indebted for the observations, examined about twenty head of cattle awaiting sale. During this operation he noted a large tabanid fly alight on a cow and attach itself to it for some five minutes. As the tabanid group had from time to time been under suspicion as a possible carrier of the disease, the cow was bailed up and examined. Mr. Rheuben, who is an expert in the manipulation of live animals, could detect no nodules in the neighbourhood of the bite. The cow happened to be sold to a New South Wales cattle dealer and in consequence it was necessary to quarantine it for some months before admission into that State. When a month had elapsed, the cow was due for dipping and the area bitten, which had previously been marked, was closely examined. A distinct nodule about the size of a grain of rice was palpable.

At a subsequent date Mr. A. E. McGown, then Veterinary Surgeon to the Department of Stock at Townsville, allowed a bullock to be set aside for experimental purposes at the Stock Experimental Station. The greatest care was taken to ascertain that there were no nodules present, several competent examiners being agreed that nothing was palpable.

Following the rains a number of tabanid flies were caught, about seventy in all, and in six of these unidentified nematodes were detected in the neighbourhood of the proboscis. The heads of these six flies were taken at once from the slides as detected and transferred to serum prepared by Mr. McGown from the blood of the bullock in question.

Further examination was then made for nodules, but the closest scrutiny could not determine the presence even of the smallest. A small incision in the shape of a pouch was made and, while depositing the heads of the six flies therein, in drawing a cork from a small glass tube with a pair of forceps, Mr. Rheuben accidentally broke off a piece of the tube which was not recovered. The pouch was sealed with collodion and healed in a few days. A month later examination showed two nodules about the size of rice grains over the area where the experiment had been made. The bullock was examined at intervals of a fortnight and one nodule came to be as large as a good-sized almond. This was subsequently dissected out by Mr. McGown's successor, Mr. J. Legg, and it was found that the piece of glass accidentally broken off was firmly embedded in the capsule. Both Mr. Legg and Mr. Rheuben were satisfied that the nodule was an ordinary onchocercal nodule.

The bullock was subsequently sold to a firm of butchers in Townsville and Mr. Rheuben, in his capacity as Meat Inspector, had an opportunity of examining the body after slaughter. In all, four worm-nests were discovered within 3.8 centimetres (one and a half inches) of the spot at which the experimental incision had been made. No other nodules were present in the animal.

At a subsequent date (August 2, 1922), while assisting several men to free a bullock bogged in a small creek, Mr. Rheuben noticed a large tabanid biting the animal and marked the spot. Examination having failed to produce evidence of any infesta-

[1] Received for publication on March 26, 1923.

tion with nodules, the bullock was set aside for a month before slaughter. On removal of the hide, to the great interest of Mr. Rheuben, who superintended the operation, there appeared at the site of the bite a coiled worm in a state of commencing encapsulation. Careful dissection allowed of the removal of a worm which, on being uncoiled, was found to be 17.8 centimetres (seven inches) in length. Mr. Legg, of the Stock Experimental Station, pronounced it on examination to be definitely *Onchocerca gibsoni*. The worm is at present preserved in the Museum of the Stock Experimental Station, Townsville.

The species of tabanid flies which were concerned in the second observation, were identified by Mr. Gerald Hill as being in the main *Tabanus germanicus* and *Tabanus aprepes*.

From information supplied by Mr. Hill,[1] based on observations made during the period October, 1919, to September, 1920, it appears that the larvæ of *Tabanus aprepes* may be generally found in all stages of development during the latter part of the wet season in Townsville (namely, from February to April inclusive) on or near the surface of clear and moderately deep pools, amongst submerged herbage near the banks, upon the submerged stems and leaves of aquatic plants of all kinds and in floating masses of algæ.

The larvæ appear to live in the water until they have reached maturity, when they migrate from the water to high ground close by and penetrate into the soil or clay to a depth of from seven to fifteen centimetres, where they remain in the larval stage, generally with head uppermost, for several months. . . . .

*Seasonal Occurrence.*—Throughout Australia, perhaps excepting the wet belts of the North Queensland coast, there is a marked seasonal occurrence of flies of this family, namely, from about October to April.

It is readily conceded that in the observations on *Onchocerca gibsoni* recorded above there is nothing to prove that actual infestation was directly due to the bite referred to in each case. Similarly, except by slaughter and examination, it could not have been demonstrated that the animal in question was free from infestation at the time of the observation and prior to the biting, though all possible steps were taken to determine such infestation with negative results.

A series of observations, however, which proceed from the detection of a definite though tiny nodule occurring after a bite to the actual detection of a worm parasite, identified as *Onchocerca gibsoni* in a stage of partial encapsulation, the approximate relation between the wide-spread distribution of the tabanid fly and of the diseases in North Queensland and common opinion which indicates a much greater prevalence in those districts in which tabanids chiefly are bred out, indicate the possibility that the relationship between the bite of the fly and the appearance of the nodule may not be merely coincidental, but may prove to be one of cause and effect.

### Reference.

[1] Hill, Gerald: "The Bionomics of *Tabanus aprepes* and Other Australian Tabanidæ," *Bulletin of Entomological Research*, Volume XII., Part I., June, 1921.

## Reports of Cases.

### DEATH FROM PROGRESSIVE EMPHYSEMA.

By C. G. McDonald, M.B. (Syd.),
*Honorary Assistant Physician, Royal Prince Alfred Hospital, Sydney.*

Although chronic emphysema is a progressive disease ending fatally, the last illness of patients suffering from this complaint is so frequently complicated by affections of other organs, notably the kidneys, that the possibility of death due to emphysema is sometimes forgotten.

W.J.B., a short, stout, thick-necked labourer and a native of Middlesex, England, came to Australia sixty years ago, at the age of three. Early in life he became a labourer, working on odd jobs. Soon he became an employee of the Kogarah Municipal Council and worked on the roads for twenty-six years. For the final eight years of his life he was employed as a main-layer by the Australian Gas-Light Company, Limited. He was twice wedded, having four children by his first and six by his second wife.

Up to twenty years ago he was slight and short, but at the age of forty he commenced to assume the corpulence of middle age. In 1901 he had his first severe illness, acute bronchitis, which caused him to expectorate blood-stained sputum. This illness had been preceded for a few years by the development of a keen taste for alcohol, chiefly beer. His surviving wife states that during his drinking bouts he frequently failed to arrive home, spending the night on some vacant allotment under the stars. Beer and a well-filled pipe, the generous handmaids of emphysema, were the two failings of this kindly, hard-working soul. But for the devoted attention of his wife, his career might have been much briefer. Afraid of pneumonia, on the least suggestion of a cold, she would put him to bed and nurse him. Yet every winter for more than twenty years he suffered from a winter cough. These attacks of bronchitis were marked by dry, paroxysmal cough, wheezing, a sense of constriction across the front of the chest and nocturnal orthopnœa. In the words of his wife, he coughed with tremendous force.

In the last four years of his life the attacks became very bad. Once he staggered home from work. His wife made a plausible but incorrect diagnosis and did not as usual go out to meet him. Her husband was not drunk this time. He was suffering from the dyspnœa of emphysema so well described by Samuel Gee. His face was purple; he reeled from giddiness and a hopeless, spasmodic cough shook his chest. On entering his home he fell unconscious to the floor. For two years prior to death he made use of a motor 'bus to take him to the train, a distance of a quarter of a mile. The gradual ascent of a long hill was too much for him. While at rest he was comfortable, but on the least effort attacks of dyspnœa came on. How he was able to work within a few months of death seems incredible. A stout heart and a cheerful disposition carry a man a long way.

When I examined him in April, 1922, he had all the signs of advanced emphysema. His chest was so ballooned that his neck seemed abnormally short. The sterno-mastoid and scalene muscles stood out like cords, even when he was at rest. The breath sounds were indistinct, but sonorous rhonchi and sibili were audible all over his chest. His wheezing and shortness of wind gave a staccato expression to his voice. The amount of expansion on forced inspiration was very small. The heart showed physiological dilatation, especially on the right side. The veins in the neck were full. The systolic blood pressure was 165 millimetres of mercury, the diastolic 100 millimetres. No abnormality of the urine was detected. The exhibition of iodide of potassium gave him temporary relief.

Early in September, 1922, there was definite cardiac failure and his life was obviously nearing its end. The apex beat could just be felt in the fifth interspace, slightly anterior to the anterior axillary line. There were no murmurs. The legs were œdematous and there was evidence of free fluid in the fat abdomen. The urine contained a moderate amount of albumin, due to the renal congestion. The large, emphysematous chest was now

motionless—no upward nor outward expansion. The work of respiration was carried on by the diaphragm alone. The attempt to lie down changed his bluish colour to an alarming purple. The poor fellow refused to remain in bed. The open window and the open door were his haunts. The anoxæmia was profound and he tried every means to feed oxygen to his tissues. About this time he was seen by Professor Mills, who confirmed the diagnosis.

On October 25 he died, fully conscious to the end. *Post mortem* the lividity of the body was extreme.

**Discussion.**

Emphysema is the result of repeated attacks of dyspnœa. This dyspnœa is mainly associated with acute exacerbations of chronic bronchitis. In dyspnœa inspiration and expiration are both forced. Now forced inspiration and forced expiration tend to produce the same result in the chest walls and the lungs. Let us first see what happens during a forced inspiration. During a forced inspiration the accessory muscles of inspiration raise and fix the upper part of the chest so as to permit the intercostal muscles to use their inspiratory action with greater power. This expansion upwards and outwards of the upper portion of the chest leads to a similar expansion of the upper portions of the lungs. The vesicles become distended with air. The lower portions of the lungs become distended also, but, as we shall presently see, this distension does not play so important a part in the production of emphysema, for, whereas the air in the inferior portions of the lungs can escape into the bronchi without much difficulty, the air in the upper portions is caught like a mouse in a trap.

After a forced inspiration comes a forced expiration or a cough which is simply a forced expiration obstructed by a partially closed glottis. The contracted *recti* raise the intra-abdominal pressure and the relaxed diaphragm is driven upwards. At the same time the chest walls are contracted or compressed. The air in the lower portions of the lungs is driven into the bronchi. When bronchitis of the larger tubes exists, plugs of mucus adherent to the walls of the larger bronchi offer obstruction to the exit of the air. Obstruction is also offered by the partially closed glottis. The air takes the line of least resistance and passes up the tubes which lead to the upper portions of the lungs. The upper and anterior portions of the lungs become distended with part of the air issuing from the lower halves of the lungs. Hence obstruction to the exit of air from the upper portions of the lungs takes place and these portions tend to become more and more distended. The trouble does not end here, for forced inspiratory movements are now necessary to draw fresh air into the over-burdened alveoli. In bronchitis and emphysema the air has little difficulty in entering the chest. The trouble is to expel it. Hence expiration is abnormally prolonged. In laryngeal diphtheria and *laryngismus stridulus*, on the other hand, there is great obstruction to the entrance of air at the glottis, the negative pressure within the chest rises to an alarming extent, the chest walls are driven in and the lower end of the sternum becomes invaginated.

Repeated attacks of bronchitis, distending the chest to its greatest capacity, finally destroy the elasticity of the chest wall and the over-burdened lungs. The chest becomes barrel-shaped and the costal cartilages rigid. Any influence interfering with the movement of the chest walls, such as a too-long-applied plaster splint, will destroy the elasticity of the chest walls and therefore of the lungs. In emphysema the loss of elasticity of the chest walls and the abolition of the normal recoil of the chest walls destroy the intrapleural negative tension. The diaphragm is the only respiratory mechanism which remains intact. As the negative pressure which exists in the normal chest facilitates the circulation of blood through the lungs, the alteration of this pressure to a positive level throws more work on to the right side of the heart and cardiac failure ultimately takes place. Moreover, as blood can now enter the chest under positive pressure only, the veins in the neck become distended with blood.

Patients afflicted with emphysema have little vital capacity, that is to say, the amount of air which they can expel by a forcible expiration after a forcible inspiration is very small. They make little impression on a spirometer. We tend to place great confidence in the diaphragm, but it shapes poorly when put to the test. The patient whose case is related above, had little expiratory power—hardly enough to extinguish a match.

Much can be done for the victim of emphysema if he consults a practitioner before his chest becomes fixed. Our aim is to increase the elasticity of his lungs and chest wall. The clothing should be light and braces should be discarded in favour of a belt. Tight collars are anathema. Graduated exercises between attacks of bronchitis promote better aeration of the lungs. Deep breathing exercises, through one or both nostrils, help to increase the vital capacity, to restore the negative pressure within the chest (by restoring the elasticity of the lungs and chest walls) and to prevent further fixation and calcification of the costal cartilages.

## A CASE OF ACUTE GANGRENOUS CHOLECYSTITIS.

By James M. Andrew, M.B., B.S. (Melb.),
*Resident Surgeon, Melbourne Hospital.*

Mrs. E.F., aged forty-nine years, was admitted to the Melbourne Hospital on November 25, 1922, complaining of acute pain in the upper part of the abdomen with vomiting of forty-eight hours' duration. The condition was diagnosed as *tabes dorsalis* ? cholecystitis. Her own private practitioner reported her condition for the forty-eight hours before admission as follows: The present attack began with abdominal pain and vomiting, which continued for about thirty-six hours. The temperature was 37.1° C. (98.8° F.) and the pulse rate 110. The abdomen was very tender and rigid over the whole epigastrium. Twelve hours later the temperature was 37.6° C. (99.8° F.), the pulse rate 124 and the respiratory rate 40. Vomiting ceased during this twelve hours and the pain was not so marked. The abdomen was still tender and rigid, most marked in the epigastrium. The lungs showed some dulness at the right base, with diminished breath sounds and a doubtful friction.

She gave the following history: Thirty years ago she had an attack of typhoid fever, from which she recovered quite well. She had only one child and had never had any miscarriages. She had been quite well since then, until twelve months ago, when she began to suffer from dull, epigastric pain after meals, chiefly in the right side of the abdomen, with occasional vomiting. Flatulence was a persistent feature of the "indigestion," as also "heartburn" and "pyrosis."

The pain in the right epigastrium at the time of admission was similar to these milder attacks, but was more severe. It was not colicky, but persistent, shooting through to the back between the shoulders and lower down, but not into the right shoulder. She had vomited about a dozen times during the thirty-six hours following the onset of the attack. She had never had any hæmatemesis or melæna to her knowledge, nor had she ever been jaundiced. Her bowels were inclined to be always constipated.

The patient was a moderately stout woman, obviously still in some pain and very ill. There were abundant sordes on the lips. Her breath was very foul and her tongue was dirty and dry. The temperature was 38.1° C. (100.6° F.), the pulse rate 116 and the respiratory rate 40. Her rapid respirations were of the sharp, jerky type, suggestive of pleural irritation. She was not jaundiced; the conjunctivæ were quite normal. The urine contained some albumin, but no sugar and no bile salt or bile pigment.

The right pupil was large, while the left one was small and pin-point; neither pupil reacted to light, but both reacted to accommodation.

The knee jerks and ankle jerks were present. There was loss of sensation in the *tendo Achillis*. Her joint sense tested in both legs was quite good.

The heart sounds were rapid and clear and the cardiac dulness was normal.

There were diminished breath sounds at the bases of both lungs, very pronounced on the right side, on which the movements of the chest wall and diaphragm were obviously limited. The breathing nowhere was tubular and percussion gave only slight impairment of resonance at the base of the right lung. There were no adventitiæ to be heard and the voice sounds were a little less distinct

at the right base. There was no friction rub in the right axilla.

Over the right epigastrium there was marked tenderess to pressure, with maximum intensity over the gall bladder itself. The abdominal wall was hyperæsthetic over the lateral and anterior distributions of the sixth, seventh, eighth and ninth thoracic segments on the right side. There was also rigidity of the upper part of the right rectus muscle. The rest of the abdomen was neither tender nor rigid and nowhere could any abnormal mass be felt. The gall bladder was not palpable.

At operation the following morning the condition discovered by Mr. Hailes, Honorary Surgeon to Out-Patients, Melbourne Hospital, to whom I am indebted for permission to report this case, was as follows:

When the abdomen was opened, in the right epigastrium, clear-coloured fluid escaped. The gall bladder was globular, distended and firmly adherent to the liver over the whole of its upper surface. The peritoneum adjacent and covering it was thick, friable and densely coated with lymph, as was also the lesser omentum, duodenum and the upper part of the right kidney. The gall bladder was gangrneous and there were several ulcerated patches on the free and hepatic surfaces; one of these was leaking into the abdomen. The gall bladder had to be partially emptied by aspiration before the pedicle with its duct and artery could be ligated. It was then removed and a large, bare area of liver was left which was oozing freely. It was impossible to cover it as there was no peritoneum left. The wound was therefore firmly packed with iodoform gauze, a drain tube inserted and the abdomen closed. When the gall bladder was opened, it was found to be half full of a mass of small stones and biliary mud, the largest stone being not greater than three millimetres in diameter.

Cultures from the gall bladder contents and the free fluid of the abdomen were made. No organism was grown from the latter, but from the former a pure culture of *Bacillus typhosus* was obtained. No other organism was present. The following week some blood was sent to the laboratory to be tested for the Widal reaction. The results were as follows:

| Bacillus. | Dilution. | | | | |
|---|---|---|---|---|---|
| | 1:10 | 1:160 | 1:320 | 1:640 | 1:1240 |
| *Bacillus typhosus* .. .. | | + | + | + | — |
| *Bacillus paratyphosus A* | — | | | | |
| *Bacillus paratyphosus B* | — | | | | |

At the same time on examination of the urine and stools no organisms were detected either microscopically or on culture. This was repeated again five days later with the same results. The patient's general condition was splendid after operation and she made an uneventful recovery in three weeks.

Locally the wound gave much trouble by continued drainage and superficial sloughing and did not entirely heal for over a month after she left hospital.

I think that the unusual features of this case are that after a lapse of thirty years the typhoid infection should have been of such virulence as to cause an acute gangrenous cholecystitis in the absence of any secondary infection.

# Reviews.

## A TEXT-BOOK OF MEDICINE.

The first edition of Taylor's "Practice of Medicine"[1] was published in 1890 and now, after occupying for thirty years one of the foremost places as a text-book for students and practitioners, appears the twelfth edition.[1]

Dr. E. P. Poulton, who has undertaken the presentation of the new edition, has retained to a large extent the features of the former editions which have made it such a complete and concise volume for the use of the student and at the same time a useful book of reference for the general practitioner. It is claimed for the work that "in a single volume the whole of medicine is represented, including such a specialty as diseases of the skin, a subject which is often omitted from text-books on medicine." The inclusion of this latter subject certainly renders the book exceedingly valuable, but in addition the range of medicine covered is complete and yet the subject matter is admirably presented and systematically arranged, though not lacking in continuity. Associated with Dr. Poulton are Dr. C. P. Symonds, who revises the section of nervous diseases, and Dr. H. W. Barker, who deals with the section of diseases of the skin.

The volume has been brought thoroughly up to date and some changes in classification have been made.

*Encepehalitis lethargica* is classified among the infectious diseases. A separate section deals with the dietetic deficiencies (beri-beri, scurvy, rickets, hunger osteo-malacia, pellagra and hunger œdema). In view of recent advances in investigation of these subjects this classification is certainly necessary.

Diseases of metabolism are held to include the diseases of the glands of internal secretion, with which is included *diabetes mellitus*. This, too, seems to be a satisfactory classification, all these diseases being regarded as essentially metabolic disorders.

Throughout the whole book there is an admirable arrangement of a short introduction to each section before the individual diseases of the section are described. Thus, under the caption "Infectious Diseases," there is a description of the nature of infection, the prevention of infection, immunity, anaphylaxis, pyrexia and its treatment. Diseases of the urinary organs are preceded by a description of the methods of examination of the kidney and bladder, the methods of examination of the urine and the estimation of the renal functions. The latest accepted methods of estimating thef unctional efficiency of organs, such as the kidneys, liver, pancreas and stomach, are presented in a practical and useful form. Indeed, the ground is so completely covered in the detailing of clinical and pathological methods, diagnosis and therapeutics, that to the student other books dealing with these separate subjects are scarcely necessary.

Interesting, precise and concise sections are those on the treatment of chronic cardiac disease, on effort syndrome, *encephalitis lethargica*, accessory food factors and the psycho-neuroses.

The classification of diseases of the nervous system, always a difficult problem, is not satisfactory. Thus, encephalitis, abscess of the brain, infantile cerebral diplegia and *paralysis agitans* are dealt with under successive headings, though their association one with another is not obvious. Progressive bulbar paralysis is included, perhaps more properly, among the diseases of the *medulla oblongata* instead of, as is usual, in association with progressive muscular atrophy and amyotrophic lateral sclerosis. It remains for some future classification of diseases of the nervous system to be as efficient as those of the other systems. The difficulty would seem to be that the classification is neither completely anatomical nor completely pathological.

The section on diseases of the skin is comprehensive and the methods of treatment are carefully detailed and should be of extreme value to the general practitioner. Such a section adds to the usefulness of the volume and is particularly welcome, since the authors of books dealing with diseases of the skin approach the subject rather from the point of view of the specialist than that of the student or general practitioner. The omission of coloured plates from this section scarcely detracts from its usefulness. Illustrations and charts are not numerous. Illustrations always enhance the value of a publication, but it would be an unreasonable demand to ask for many pictures and diagrams in this book, for this would entail a further considerable increase in size and an alteration of price. The standard of those presented is excellent.

This text-book, "a splendid monument to the industry and knowledge of its author," is indeed in its revised form a comprehensive and concise presentation of the whole range of medicine.

[1] "Taylor's Practice of Medicine," by E. P. Poulton, M.A., M.D. (Oxon.), F.R.C.P. (Lond.), with the assistance of C. Putnam Symonds, M.A., M.D. (Oxon.), M.R.C.P. (Lond.), and H. W. Barber, M.A., M.B. (Camb.), F.R.C.P. (Lond.); Twelfth Edition; 1922. London: J. & A. Churchill; Royal 8vo., pp. 980, with 24 plates and 87 text figures. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, APRIL 21, 1923.

## Typhus Fever.

The continued appearance of an acute infective disease simulating typhus fever in many respects in South Australia raises some important issues. In the first place, it is necessary to ascertain whether this disease is typhus fever or a hitherto undescribed infection. In the second place, it is a matter of practical importance to determine the method of spread of the infection, since the prophylaxis necessarily depends on this. The third question involves an examination of the significance of poly-agglutination. Dr. F. S. Hone's admirable description of the two series of infections and his cautious, yet skilful discussion of the epidemiological problems surrounding these infections should be studied by every practitioner in the Commonwealth, since it may transpire that the extent of the disease is wider than has been demonstrated.

The clinical similarity of the affection with typhus fever must be apparent to everyone who has had experience of the latter. This is strengthened by the fact that the serum of nearly all the patients agglutinated *Bacillus proteus X 19,* an organism isolated from the blood of persons suffering from typhus fever in 1916 by E. Weil and A. Felix in considerable dilution. Recent researches, however, have led us to believe that typhus fever is invariably transmitted by the body or head louse, while the South Australian disease is certainly not propagated in this manner. If there is ground for belief that typhus fever cannot be carried from one person to another, except by way of the bites of *Pediculus corporis* or *Pediculus capitis,* then this disease is not typhus. The history of the investigation into the ætiology and pathology of typhus fever is highly interesting and from the information contained in it there would seem to be an indication of the solution of this difficult problem.

As recently as 1908 it was held that the virus of typhus fever is given off in the expired air and in the exhalations from the patient's body. Muchison's view of spontaneous generation of the infecting organism had been abandoned by that time. In 1909 Charles Nicolle demonstrated that the disease could be transferred to apes and the lower monkeys by the injection of the blood of a patient. In 1910 H. T. Ricketts and R. M. Wilder discovered small bacillus-like bodies in the blood of patients suffering from the Mexican form of typhus. These bodies, which were regarded as bacteria, were found in small numbers in the blood, but they were very numerous in the fæces and intestinal wall of lice that had been fed to persons with typhus fever. At first this discovery did not attract much attention. Nicolle endeavoured to show that the virus was a filter passer, since he succeeded in 1911 in inducing the disease in monkeys and guinea-pigs by injecting the blood of infected patients after it had been passed through a Berkefeld filter. It is probable that the filter was inefficient. In the meantime the fact had been noted that persons affected with this disease were always infested with lice or bore marks of louse bites. Nicolle utilized this observation and produced experimental typhus in monkeys and in man through the intermediation of *Pediculi.* He claimed that the virus was introduced by the act of biting. At the beginning of the war very little was known concerning the virus of typhus and its transmission from man to man. The enormous spread of the fever, especially in Russia and Poland and in the south-eastern quarter of Europe, provided an opportunity and a stimulus for further study. In 1914 Rocha-Lima, Prowazek and Sikora rediscovered the organism that Ricketts and Wilder had described and, being more acquainted with protozoa, they recognized that it was not a bacterium, but a protozoon. Rocha-Lima renamed the organism *Rickettsia prowazeki* and classified it among the *Protozoa strongyloplasmata.* This protozoon has not yet been cultivated artificially. It is found in lice that have fed on persons suffering from typhus fever from the fifth to the eighth day and in guinea-pigs that had been subjected to the bites of infected lice. Rocha-Lima found that the lice usually remained infected for the rest of their lives. The protozoon has been found in lice that have not had

an opportunity of feeding on typhus patients. At first this fact was thought to be incompatible with the view that it was the causal organism of typhus. It was, however, shown by J. A. Arkwright and others that another form of *Rickettsia* could be recovered by means of lice from the blood of soldiers affected with trench fever. Rocha-Lima also held that there were several varieties of *Rickettsia*. A considerable amount of work has been carried out in connexion with this subject and it may now be held that *Rickettsia prowazeki* is the causal organism of typhus. Another form of *Rickettsia* has been discovered in association with Rocky Mountain fever. This organism is conveyed by means of ticks. It is necessary to adduce further evidence in support of this contention, such as the immunological reactions of the patient's serum to emulsions of the virus, but this work is difficult, owing to the fact that the virus has not yet been cultured *in vitro*. The work is not only difficult; it is very dangerous. Ricketts, Prowazek and Schüssler have sacrificed their lives in this study.

In 1909 Horiuchi noted some motile organisms in the urine of persons suffering from typhus fever. Presuming that these bacteria might be the causal organisms of the affection, he tested the power of the patient's serum to agglutinate emulsions. He found that agglutination occurred even in high dilutions. In the following year W. J. Wilson made the same observation. He recognized, however, that the organism found in the urine was not the virus of the disease. Six years later E. Weil and A. Felix isolated a member of the *proteus* group from the urine of a patient with typhus fever. They found that while the serum of patients suffering from this disease agglutinated this organism in high dilution, the serum of persons not suffering from typhus fever failed to do so. They called this bacterium *Bacillus proteus X 19*. Whatever views were held a few years ago concerning the *proteus* organisms, it is now known that they have nothing to do with the causation of the disease. Weil and Felix were struck by the constancy of the reaction between the serum of typhus patients and the *proteus* organisms and therefore devised a test which is now known as the Weil-Felix test. It is claimed that if a suspect serum diluted fifty times or more with saline solution agglutinates *Bacillus proteus X 19* in suspension, the illness is typhus fever. A very large number of patients have been subjected to this test and it has been found that the serum of nearly every patient with typhus fever causes agglutinations in dilutions of one in fifty or less (the reaction has been observed in as low a dilution as one in thirty thousand). The serum of persons suffering from other conditions does not induce this reaction. The technique has been modified by W. James Wilson and others and the test is now regarded as a reliable one in this specific disease. It should be pointed out that the serum of persons suffering from typhus fever agglutinates many other organisms, some of which are not related to the *proteus* group. It would seem that the serum contains what are called poly-agglutinins. Moreover, it has been demonstrated that the urine of a patient whose serum reacts well with the *proteus* bacillus, usually does not harbour this bacterium. The organism is therefore not a secondary invader in the ordinary sense of that term, nor does it exist in symbiosis with the virus of typhus fever. All that can be said at present is that the serum of persons with typhus fever contains agglutinins which react with a large and diverse group of bacteria. The infection in South Australia has this characteristic in common with typhus fever. No other known illness shares it. It must therefore be admitted that there is strong presumptive evidence of identity.

Is there any method that would yield an unambiguous answer to the question of the true nature of the South Australian infection? Those best able to form a reliable opinion have arrived at the conclusion that *Rickettsia prowazeki* is the virus of typhus fever. It has been demonstrated that if lice, bred in the laboratory and fed to healthy persons or animals, be allowed to bite persons infected with typhus, this protozoon can be recovered and recognized in the lice. Moreover, lice fed to patients with typhus fever become capable of producing a typical infection in monkeys and guinea-pigs. The differentiation between *Rickettsia prowazeki* and *Rickettsia pediculi* or other varieties appears to be difficult. This difficulty could be overcome by control experiments with lice not fed on patients' blood. It should be possible to conduct both experi-

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ments in connexion with the patients in South Australia and to obtain evidence of considerable value for or against the hypothesis that this infection is typhus fever. In the meanwhile Dr. Hone's invitation to medical practitioners to watch for infections of this nature should be taken very seriously.

## THE QUEENSLAND MATERNITY HOSPITAL SCHEME.

During the last session of the Queensland Parliament a measure known as the *Maternity Act* was passed for the purpose of establishing maternity hospitals, hospitals for the treatment of diseases peculiar to women, ante-natal and baby clinics and training schools for nurses in midwifery and infant welfare. These objects are so admirable that the general support of all sections of the community for the measure is assured. The programme is a very ambitious and enterprising one and at first sight it might be questioned whether this measure would not share the fate of many other far-reaching Acts of Parliament, namely, of reposing unremembered and unnoticed in the Statute Book. It has been impossible, owing to the limitation of our space, to deal with this piece of legislation from the time when it emerged from the draughtsman as a Bill until it obtained sanction. The value of this Act necessarily must depend on the method of application of the provisions and on the machinery that is created for its several purposes. The Home Secretary has recently taken the public into his confidence and has elaborated his scheme for carrying into effect these provisions. It is announced that the approval of the Cabinet has been given to the scheme. It involves an expenditure of about £225,000. We refrain from expressing any opinion in regard to the morality of the means adopted to provide this money. In plain English, it is being raised out of the foibles of the masses. The "Golden Casket" lottery, playing as it does with the gambling propensities of the average man, is an inexhaustible mine. Hospitals would be established in many places on the understanding that the local community would undertake to maintain the institution with the assistance of a Government subsidy. The new maternity hospital would be established on the district system. In the first place, the needs of "out-back" places would receive consideration. Then small towns hitherto without maternity hospitals would be offered the chance and lastly it is proposed to amplify the provisions in places where the existing arrangements are insufficient for the population of the district. Hospitals for diseases of women and ante-natal and baby clinics would be established only in the larger centres. It is proposed to train the nurses engaged at the country maternity hospitals in ante-natal and infant welfare work, so that preventive measures might be available for women living out of reach of the larger towns. The scheme further embraces provision for the training of nurses at special centres or schools. A formidable list of places is given at which maternity wards are to be established in association with existing hospitals. In some isolated places cottage maternity hospitals are to be erected. In the next place, sums varying between £2,500 and £10,000 are to be set aside for the provision of adequate institutional accommodation in the ten largest centres in Queensland. It appears that the committees of the hospitals will be permitted to make charges for maintenance, but it is specifically set out that no person is to be refused the benefits of the maternity hospitals because they cannot pay the fees for maintenance. There is a significant addition to this provision. The benefits may not be refused "on account of any other cause." This would mean that neither poverty nor wealth is to prevent women from using these institutions. The prescribed charges are to be maximal charges; reduction of fees may be made to persons of limited means. The medical officers of the institutions are to be permitted to attend to their private patients in these maternity hospitals, provided that the admission of these patients does not lead to the exclusion of "patients for whom the hospitals are required." The payment of the maintenance charges by private patients must be safeguarded. It appears that under the ordinary rules of country hospitals in Queensland the medical officers are permitted to treat their private patients in the hospitals. It also appears that no alteration in practice will be introduced by the establishment of maternity hospitpals and maternity wards in existing hospitals. The Hospital Sub-Committee of the Queensland

Branch of the British Medical Association is of opinion that the proposed arrangements are satisfactory and that the interests of medical officers to these institutions will be protected. The Sub-Committee has further considered the question of the arrangements for the nursing of the patients in the maternity wards of general hospitals. It seems that, as the same conditions obtain at present at many private hospitals, no insuperable difficulty need be anticipated.

From the foregoing it will be gathered that women of all classes are to be given the great advantage of institutional treatment during child-birth. In the cities and larger towns the accommodation in the public hospitals will be limited. Since the less well-to-do are to have preference, those who can afford to pay, will continue to seek admission to private hospitals. In the small towns and country districts an endeavour will be made to provide for all. Private patients will be placed in these hospitals by their own medical attendants and will be treated by them. The arrangements for the ante-natal care of women are less clearly defined. In the larger centres clinics are to be established. If competent medical officers are appointed to undertake the examination of the patients and to institute prophylactic treatment when necessary, nothing but good can arise. Private practitioners will continue as heretofore to look after their patients in their own homes during the period of the pregnancy. In the country districts, it would seem as if it were contemplated that the nurse is to be responsible for this ante-natal control. Such care is obviously insufficient and may be dangerous unless properly supervised. In places far removed from medical aid it might be advisable to accept this expedient as the only alternative to no aid. But where a responsible medical practitioner is available, the nurse in the maternity hospital should be prevented from playing the part of the doctor.

The question of the baby clinic also needs careful watching. Experience has shown that the baby clinic nurse is only too apt to pose as a physician, with the result that the mothers are given worthless advice, to the immense detriment of their infants. A well-trained clinic nurse will act under instructions from a practitioner skilled in the care of infants. She will keep careful records of the progress of the babies, influence the mothers to continue breast feeding until the babies are nine months of age and refer the babies to the doctor when the first signs of disturbed health are detected. The reckless nurse who treats baby on her own responsibility until the matter becomes serious, should not be permitted to remain in the clinic.

There is much that will need supervision and watching in this interesting scheme of the Home Secretary of Queensland.

## Current Comment.

### DENTAL CARIES.

In 1913 O. Walkhoff wrote that there is scarcely a tooth of civilized man in which some defects of structure cannot be found in the development of its elements, either in their size and arrangement or in regard to their calcification. The wholesale examination of the teeth of school children, which can obviously bring to light only gross defects, reveals an alarmingly high incidence of decayed teeth. Every medical and dental practitioner realizes the disconcerting prevalence of caries in the teeth of adults. There is no need to adopt an alarmist attitude on account of high incidence of pyorrhœa when this indictment of the ubiquity of actual dental disease thrusts itself on public attention. The first impression is that this defect is a penalty of our so-called civilization, but this is apparently not the case. Walkhoff has found that the teeth of the large anthropoid apes and especially the enamel show the same imperfections as the teeth of man. The problem of healthy teeth thus becomes much more difficult. In view of the importance of good, clean teeth to the nation, the Medical Research Council, after consultation with the British Ministry of Health, appointed a committee to investigate the causes of dental disease. The members of this committee are Professor W. D. Halliburton, F.R.S., Dr. Norman G. Bennett, Dr. Leonard Colebrook, Dr. J. M. Hamill, Sir Arthur Keith, F.R.S., Mr. P. P. Laidlaw, Mrs. Edward Mellanby, Dr. J. H. Mummery and Dr. C. J. Thomas. The Council announces in a preface to Dr. J. H. Mummery's monograph on the structure of teeth in relation to dental disease[1] that the reports of the work will be published from time to time and that the Committee will form their conclusions at a later date when all the data have been collected.

Dr. Mummery first of all examines the question of the relationship of the variations in composition and structure of enamel to caries. He shows that a lively controversy has taken place in regard to the evidence of organic structure in enamel. The prob-

[1] "The Structure of Teeth in Relation to Dental Disease" (First Report of the Committee for the Investigation of the Causes of Dental Disease), by J. Howard Mummery, C.B.E., D.Sc., M.R.C.S., L.D.S.; Special Report Series, No. 70, Medical Research Council of the Privy Council; 1922.

lem has been attacked both from the point of view of the chemical composition of enamel and from that of its histological characters. Tomes found no trace of organic matter when enamel was analysed by the ignition method. On the other hand, Lovatt Evans detected between one and two parts of carbon in each hundred parts of enamel. The histological evidence is more reliable. It appears that certain stains can penetrate enamel through the so-called enamel tubes, which are spaces between the prisms containing uncalcified tissue. The stain (fuchsin in alcoholic solution) is allowed to penetrate the tooth by capillary attraction from the end of a root. In teeth stained in this way the spindles or spaces in the enamel matrix are seen to be in direct communication with the dentinal tubes. At times they are clear and stain deeply, while elsewhere they are opaque and unstained. Radiating bundles of what appear to be fibres are seen at intervals at the margin of the dentine and enamel. These are partly stained and partly unstained. They interlace and cross one another and in consequence it is difficult to determine whether they are fibres or merely stained interprismatic spaces. Some evidence has been adduced which gives weight to the contention that the enamel of the erupted tooth is incomplete and is capable of further consolidation. Dr. Mummery has demonstrated that this process takes place in certain fishes (*Sargus nocturnus—Sparidæ*). This question, however, is now held to be of secondary importance, since the penetrability of the enamel is a more potent factor in the ætiology of caries than are defects of calcification.

Many attempts have been made to produce caries artificially. As long ago as 1867 E. Margitot endeavoured to prove that caries is a purely chemical process. He was convinced that the products of acid fermentation in the saliva or active substances introduced into the mouth, caused the decay. In those days ivory dentures were in use. Later porcelain teeth have been substituted for this purpose. It had been noted that in some instances the dentures underwent rapid destruction in the mouth. At times true cavities formed in the ivory, filled with detritus yielding an acid reaction. Margitot carried out many interesting experiments with extracted teeth and found that teeth immersed in sugar, lactic acid and butryric acid solutions underwent changes indistinguishable from caries, but that no changes resulted if the pabulum were alkaline. It appears, however, that Margitot did not take bacterial action into account. Miller and Underwood immersed teeth in a mixture of bread, saliva and meat and incubated the vessel at body temperature. Since no changes took place, they concluded that artificial decay could not be induced, because the bacteria lost their specific power when outside the mouth. Miller soon realized the defect of this view. He took the precaution to change the bread and saliva mixture frequently, so that it was not allowed to become alkaline. Miller found that Nasymth's membrane offered a distinct resistance to the action of the acid and that a thin surface layer of the enamel was also less readily attacked by the acid than the deeper portions. He held the view that the process was dependent on an initial decalcification of the enamel and dentine by the acids derived from carbo-hydrate fermentation. The fermentative bacteria then entered between the prisms of the enamel and traversed the tubes of the dentine. They proliferated in the dental tubes. He further postulated an enzyme which he supposed acted on the organic matrix of the enamel. In many preparations Miller's zone of decalcification is plainly visible, as are the liquefaction foci and the invasion of the dentinal tubes by bacteria. The author finds some reason for assuming that mucin plays a small part in the production of decay. He is convinced that the translucent zone in the dentine is an indication of a vital reaction on the part of the pulp. It serves as a barrier to the penetration of the bacteria.

In the third place Dr. Mummery deals with the subject of arrested caries, a phenomenon that is occasionally encountered. It affects the deciduous molars more commonly than the permanent teeth. Hypoplastic teeth seem to be more frequently the site of arrest of the process than well-developed teeth. It is stated to be due to mechanical causes as well as to biological processes. It appears that the exposed surfaces in which arrest of caries has occurred, provide a polished appearance; the enamel is worn and rounded and the dentine is pigmented. If the carious process arises in deep fissures arrest is unlikely to occur, but when it starts superficially in teeth of good structure, it may be expected. The reason for this is readily understood when the arrangement of the prisms is studied at the cusps of the molars and premolars. When caries attacks teeth with living pulp, a clear zone of dentine is seen between the pulp and the affected area. This zone, as mentioned above, represents a reaction on the part of the pulp in an effort to stem the advancing bacteria and thus to preserve the sensitive tissue. It is the result of irritation and in consequence is associated with the deposition of secondary dentine. Both the zone of translucency and secondary dentine are found in teeth in which the carious process is arrested. Dr. Mummery points out that of itself this area of translucency is incapable as a rule of preventing the spread of the caries and the final destruction of the tooth. When assisted by mechanical means and especially when the alterations accompanying the pigmention of the outer zone of the dentine are superadded, it may effectually prevent further encroachment of the process. Hitherto no chemical means have been discovered to imitate this process.

It would be futile and unwise to endeavour to subject these observations and deductions to critical analysis at this stage. The presentation of the facts is admirable and the facts themselves are obviously of importance. Further information concerning the physical, chemical and biological processes involved in the causation of dental disease will be recorded by the Committee. The result of this work will be awaited with interest and not a little anticipation.

# Abstracts from Current Medical Literature.

## PÆDIATRICS.

### A New Eruptive Fever.

A. M. Stevens and F. C. Johnson (*American Journal of Diseases of Children*, December, 1922) describe two instances of an extraordinary, generalized eruption with continued fever, inflamed buccal mucosa and severe purulent conjunctivitis. The condition was so unusual and so unlike anything previously observed that pains were taken to get as many expert opinions as possible from men of wide clinical experience. A search in the literature with reference to eruptive fevers failed to reveal any condition resembling it. Both patients were boys, one aged seven, the other eight, coming from widely separated parts of New York and with no possibility of previous contact. Both patients manifested a purulent conjunctivitis, in the elder going on to panophthalmitis and total loss of vision and in the younger responding to treatment by leaving a severe corneal scar. Examination of the pus showed the presence of pyogenic organisms; no gonococci were found. A high and continuous fever was present in both instances. This was explainable in the elder boy by a lobar pneumonia, but in the younger it was without apparent cause other than the skin condition. The eruption showed certain characteristics, identical in each instance. The onset was with fever, the rash appearing on the back of the neck and chest, spreading to the face, arms and legs during a period of about eighteen days, the last lesion to appear being on the soles and palms. At this time resolution of the first lesions began. The eruption consisted of oval, dark red to purplish macules, separated by normal areas of skin. These became in a few days raised, firm papules of brownish purple, from 0.5 to 2 centimetres in the longest diameter, without areola. There were no subjective symptoms of pain or itching at any time. A few of the largest spots showed a yellow, dry, necrotic centre. No pustules or vesicles were to be seen. The scalp was at all times free of lesions; but the mouth and lips were intensely sore and inflamed. In the younger boy bullæ were noted in the mouth at the end of the first week. After the third week resolution began in the order of appearance of the lesions. This consisted of a shrinking of the macule to a horny oval of dark brown colour, with raised, papery edges. From the fourth week these scales dropped off, leaving a faint pigmented area without pitting or scarring. Fall of temperature coincided with the period of resolution of the skin lesions. The diagnosis of the condition was left an open question. Careful consideration was given to the possibility of various known ailments, such as drug or food poisoning, syphilis, pemphigus, hæmorrhagic measles, generalized sepsis and *erythema multiforme*, but without result. The authors point out that the picture presented was a striking one; a syndrome of dramatic onset, with fever, conjunctivitis and cutaneous eruption. The child was prostrated, the mouth and tongue were inflamed and raw, the eyelids were swollen and pus streamed from the eyes. There was a course of three or more weeks of high fever, with leucopenia. It suggested an infectious disease of unknown origin.

### Protein Sensitization.

J. Francis Ward (*Archives of Pediatrics*, November, 1922) discusses protein sensitization as the common factor in whooping cough, asthma, hay fever and influenza. The symptoms and pathology of whooping cough are the same as those of hay fever. In whooping cough it is the upper respiratory tract which is affected, the trachea and glottis. In influenza the bronchi are the seat of disease. The majority of whooping cough infections occur in the warmer months, when plant pollination is at its height. The epidemics of influenza, however, have started in the late autumn and are undoubtedly due to pollens, the proteins of which form a virulent poison. The poisons irritate the peripheral nerve endings, causing itching and cough. In whooping cough the œdema extends to the Eustachian tube and produces a sensation of tenseness. The glottis becomes œdematous and causes the characteristic whoop. The whoop is more noticeable at night and this fact brings out the connexion between the disease and the emanations from animal dust protein. The chicken, duck and goose feathers contained in the pillow are themselves a potent cause of protein poisoning. Whooping cough is a forerunner of a later hay fever and asthma. In both whooping cough and influenza streptococci are found in great numbers, the soil having been prepared for them by protein poisoning. Whooping cough, asthma, hay fever and influenza are caused by pollens, but the focus of infection and the specific pollen are the variable factors depending on individual idiosyncrasy. The flora of different localities or different countries appears to govern both the type of infection and the season of its appearance. Allergy and anaphylaxis are present in the majority of these diseases. Allergy indicates the congenital form of hyper-sensibility to pollen protein. Anaphylaxis indicates sensitization of an allergic subject resulting from exposure to pollen protein in excess of inherited resistance. In addition to its high mortality rate, whooping cough produces sensitization that can be dormant for years. Children should be carefully treated during an attack and afterwards kept so robust that their natural powers of resistance form an effective barrier to all the so-called infectious diseases.

### Congenital Heart Disease.

E. Bellingham-Smith (*The Practitioner*, November, 1922) points out that the prognosis of congenital heart disease is not always as grave as it is stated to be. He divides his patients into four groups: (*i.*) those in whom the disease is discovered *post mortem*, (*ii.*) children attending hospital, (*iii.*) children in whom the disease is discovered during routine examination at school, (*iv.*) adults. Children in whom the disease was discovered *post mortem* were found to be nearly all under two years of age. When the condition requires treatment before two years of age, it will in the majority end fatally in infancy or early childhood. The prognosis in children requiring medical attention after two years of age and before eight is almost as bad and though the immediate mortality is not high, as in the previous group, few survive to early adult life. The prognosis in groups (*ii.*) and (*iv.*) cannot be founded with any accuracy on physical signs, but must be largely deduced from the symptoms present, namely, wasting, ashen pallor, extreme cyanosis, convulsions. These are of grave significance, while good bodily development and absence of serious symptoms make the outlook more hopeful. After eight years of age the general conditions will still play a large part in the prognosis, but the physical signs at and after this age are generally of a moderately well defined type. A prognosis may then be made of a more or less favourable character, according to the lesion present. There are many patients suffering from congenital heart disease who have never called for and may never require any medical treatment. Consequently any prognosis founded solely on hospital experience is fallacious in that it tends to attach too high a mortality to congenital heart disease as a whole.

### Retarded Mentality and Organo-Therapy.

M. J. Konikow (*Endocrinology*, March, 1922) describes the case of a girl, aged fourteen years, whose parents were healthy and whose birth had been normal. She was tall for her age and weight, but no other physical abnormality was present. Mentally she was very backward. Treatment consisted in the administration of capsules three times a day containing: Pituitary gland (anterior lobe), 0.12 gramme (two grains); thymus gland, 0.06 gramme (one grain); thyreoid gland, 0.005 gramme (one-twelfth of a grain); compound calcium phosphate to 0.3 gramme (five grains). Considerable mental improvement resulted, as shown by her school record. Renewed retardation occurred when treatment was temporarily interrupted.

## ORTHOPÆDIC SURGERY.

### Injury to the Spinal Cord in Breech Extraction.

Bronson Crothers (*The American Journal of the Medical Sciences*, January, 1923) draws attention to the possibility of injury of the spinal cord in breech extraction as an important cause of fœtal death and of paraplegia in childhood. Injuries of the spinal cord in these circumstances are fre-

quently discovered by pathologists, but are rarely reported by obstetricians and disability from such accidents is largely recognized by those who see children later in life. The obvious inference is that serious damage to the cord during delivery is inconsistent with prolonged life. The influence of Bastian may have had a good deal to do with non-recognition of the condition, because, according to the observations, trans-section of the cord results in complete loss of reflex activity in the segment below. The work of Head and Riddoch has shown that this view is false and that reflex activity returns in the human cord after the preliminary shock has passed off. The author reviews the literature of the subject and comes to the conclusion that: (*i.*) Cord lesions as a subsequence of breech extractions are not infrequent. (*ii.*) They have only been recognized when accompanied by flaccid paralysis and anæsthesia. (*iii.*) In most of the instances reported the babies do not live for any length of time. (*iv.*) In the light of the work of Head and Riddoch it seems extremely likely that such babies should show high degrees of reflex excitability, provided the lesion is not high enough to involve the medulla or the phrenic nerves nor low enough to destroy the lumbar enlargement. Under favourable circumstances, indefinite existence seems quite possible. The details of five instances which came under the author's observation are given. In all of these crippling apparently depended upon injuries of the spinal cord during a breech extraction. Four of these children showed conclusive evidence of a practically complete trans-section. One child showed evidence of an incomplete lesion. She had a flaccid paralysis dependent upon destroyed cells and lower spasticity from tract involvement, sensory disturbances and sphincter disorders proportionate to the involvement of cells and tracts. From a theoretical consideration of the forces at work in extractions it seems justifiable to suggest that traction on the cord, when combined with suprapubic pressure or uterine contractions acting on the fœtal head, may cause collapse or death from herniation of the *medulla oblongata* through the *foramen magnum*.

### Synovectomy in Chronic Infective Arthritis.

P. P. SWETT (*Journal of Bone and Joint Surgery*, January, 1923) has performed synovectomy fifteen times on eight patients suffering from chronic infective arthritis. The operation itself consisted in opening the joint at the site of greatest thickening and effusion and then with scissors and forceps dissecting out all the diseased inner layers of synovial membrane down to healthy tissue. The joints were all closed without drainage and with no fixation. The theoretical considerations that led the author to carry out this treatment were gradually formulated. His first object was simply to remove mechanical obstruction to joint movement after the septic foci of infection had been removed. The second theory was that the hypertrophied synovial membrane and the organized exudate actually contained organisms which prolonged the process within the joint. A third consideration arose from the work of Pemberton, who showed a constant sub-oxidation process in patients suffering from arthritis. Synovectomy restores function and thus indirectly provides the necessary metabolic stimulant. The exterior of the joints operated upon showed thickened and dense synovial membrane with large villous tabs or fringes or a pannus extending over the articular surfaces. Excessive synovial fluid was present, but only in one instance was there ulceration of the joint cartilage. The pathological reports showed that the tissue was the seat of chronic inflammatory changes, while cultures in every instance failed to produce growth of bacteria. Micro-organisms were found in the stained specimens taken from one patient. The author concludes that this operation cannot take the place of other forms of treatment and in every instance preliminary care should be taken to eliminate the original foci of infection.

### Fractures of the Shaft of the Femur.

E. L. ELIASON (*Surgery, Gynecology and Obstetrics*, October, 1922) emphasizes the importance of both internal and external fixation in fractures of the femur. Internal fixation is accomplished by means of a Sherman plate. At operation a fenestrated drainage tube is placed in the dependent part of the wound, brought through the dressings and covered with a pill box top to mark its position. The pill box and tube are removed forty-eight hours after operation. Some means of skeletal traction is also applied at the time of operation to the lower end of the femur and the patient is then transferred to a modified Bradford frame. To provide an attachment for extension a loop of piping is attached to the bottom of the frame in a plane at right angles to the general surface of the frame. External fixation of plaster of Paris is then applied with the hip and knee in the flexed position. Great importance is attached to this position, since the muscles attached to the femur are in a state of equilibrium. In fractures of the upper half of the femur it is advisable to abduct the limb as well as to rotate internally the lower fragment. The plaster is open in the region of the knee to allow extension to be applied and a spring balance is used to measure the amount of extension. For twenty-four hours 5.4 to 9 kilograms (twelve to twenty pounds) extension are used, but this is gradually reduced to two-thirds the quantity.

### Muscle Conservation in Paralytic Deformities.

P. W. ROBERTS (*Journal of Bone and Joint Surgery*, January, 1923) makes a plea for a conservative attitude in dealing with apparently paralysed muscles in foot deformities. He cites three instances illustrating the return of function in apparently hopeless muscles when strain was removed and normal length of the affected muscles was restored. As a result of observations in a large number of patients the author has formulated two working principles. In the first place, he holds that a muscle which does not functionate after an attack of polio-myelitis, is not of necessity permanently paralysed. Secondly, he concludes that when a group of muscles is supplied by the same terminal nerve, no member of the group is likely to be completely paralysed if the others have gained approximately normal power. The application of this principle has given rise to a very satisfactory procedure in dealing with the valgus deformity following paralysis of the *tibialis anterior* muscle. The common extensor muscle of the toes is used to reinforce the *tibialis anterior* muscle, the tendon of which is shortened at the same time. The calcaneous deformity offers a more difficult problem in treatment, on account of the fact that the gastrocnemius and soleus muscles are so much larger than any available reinforcing muscle. Return of power in the calf muscles after astragalectomy is practically a constant finding when there is active control of the flexor of the toes.

### Osteoma of the Cervical Spine.

M. S. HENDERSON (*Journal of Bone and Joint Surgery*, July, 1922) reports a case of osteoma of the cervical part of the spine. The onset of symptoms was moderately sudden. The patient, a boy, aged ten years, complained of pain in the posterior part of the neck. Sudden and jerky movements aggravated the pain which became continuous and severe after three days. A swelling was noticed in the region of the pain. His temperature varied from 37.2° C. to 38.4° C. (99° F. to 101° F.). When examined at the Mayo Clinic the patient had already undergone two operations and at the second one a hard tumour had been encountered. Specimens removed were found to consist of non-malignant fibrous tissue with occasional islands of bone. When seen by the author the patient had a globular tumour in the right cervical region and an X-ray examination showed a mass apparently attached to the third cervical vertebra. His head was bent forward so that the chin was practically on the chest and he was unable to look directly forward. At operation a circular tumour about five centimetres in diameter was outlined. It was attached to the spinous and lateral processes of the third cervical vertebra. After removal the tumour was found to consist of bone and fibrous tissue. No cartilage cells were seen. The author considers that the tumour was a very unusual one and that it should be classified as an osteoma. The origin was probably infective, but observations on this point could not be made. Enlarged tonsils were found and tonsillectomy was advised.

# Special Abstract.

## THE TOXICITY OF ARSENO-BENZOL PREPARATIONS.

The Salvarsan Committee of the Medical Research Council has issued a report on its investigations into the toxic effects following the employment of arseno-benzol preparations.[1] The members of the Salvarsan Committee are as follows: Sir Humphry D. Rolleston (Chairman), Sir Frederick Andrewes, F.R.S., Professor William Bullock, F.R.S., Dr. H. H. Dale, F.R.S., Brevet-Colonel L. W. Harrison, Dr. J. W. McNee, Major C. F. White and Dr. F. J. H. Coutts (Secretary). In compiling this report the Commitee has made an extensive survey of the literature on the subject. Free use has been made of statistics obtained as a result of the use of "Salvarsan" and its substitutes in the Army. In addition to this, the histories of many unpublished fatalities have been communicated to the Committee. The literature of other countries has been studied and a critical analysis of inquiries in these countries has been undertaken. The compilers of the report point out that many records are valueless on account of their incompleteness in regard to *post mortem* examinations.

### Incidence.

In determining the frequency of some of the ill-effects some difficulty is experienced. Many observers include as ill-effects conditions of a trivial nature which are quite disregarded by others. Again some observers in Germany, in drawing up the report of an inquiry, evidently thought that deaths from acute yellow atrophy of the liver and from acute dermatitis were in no way connected with the arseno-benzol treatment. For these reasons the records obtained from various sources are not strictly comparable. The statistics from several military hospitals are tabulated. In one military hospital ("A") 4,887 patients with syphilis were treated by "Salvarsan" or "Neo-Salvarsan." One hundred and twenty-six of these suffered from ill-effects of a more or less serious nature. There were three deaths from dermatitis. In a second military hospital ("B") 18,500 patients with syphilis were treated by 125,000 injections of "Salvarsan" and "Neo-Salvarsan." Ill-effects of varying severity occurred in two hundred and fifty instances. Ten patients died, one from heart failure following urticaria, one from mild erythema, six from severe dermatitis, one from dermatitis and jaundice and one patient who had mild jaundice, died from empyema and valvular disease of the heart. In a third military hospital ("C") 9,758 patients were treated by injections of "Salvarsan" and "Neo-Salvarsan." Among these patients twenty-six deaths occurred. Seven of these deaths were attributed to the arseno-benzol compounds; five were due to dermatitis and one to acute arsenical poisoning. It was thought probable that the drug may have had causative relation to the remaining nineteen deaths. It is pointed out that in Hospital "C" the course of treatment was more intensive. A separate table is drawn up to show the composite figures for four military hospitals, two in France and two in England. In the four units 29,946 patients were treated for syphilis. Of these, 480 gave evidence of complications. The number of deaths is not stated, but an analysis of the number of complications shows that one hundred and eighty-seven patients suffered from jaundice, one hundred and eighty-two from dermatitis, sixteen from erythema, eighty-one from urticaria and fourteen from cerebral symptoms.

In regard to the treatment of the civil population, it was shown by returns from the civil treatment centres in England and Wales during 1920 that 77,645 patients suffering from syphilis received 298,011 injections of arseno-benzol compounds, that is, less than four injections per patient. Seventy-seven of these patients gave evidence of ill-effects and ten of them died. It is pointed out that the number of patients affected is smaller than in the military hospitals. This is probably due partly to a failure to report ill-effects in civil practice and partly to the fact that the number of injections per patient was smaller. Of the ten patients who died, three had suffered from severe jaundice, three had had severe dermatitis, two had given evidence of cerebral symptoms, one had developed a Herxheimer crisis and in regard to the last patient no particulars are available.

The Committee investigated the summary of two German reports on the use of "Salvarsan" and its ill-effects. The first of these related to an inquiry held in 1914, which was the outcome of opposition to the new forms of treatment. In reply to a *questionnaire* statistics were made available in regard to 288,942 injections of "Salvarsan" given to 74,018 patients. About 22,000 of the injections were either subcutaneous or intra-muscular. Necrosis occurred in 455 instances and the number of neuro-recurrences was 168. It is generally recognized that these are the results of syphilis and not of its treatment. Twelve deaths were regarded as being certainly due to "Salvarsan." *Post mortem* examination in one-half of these revealed the presence of *encephalitis hæmorrhagica*. Instances of dermatitis and acute atrophy of the liver are not included. The German inquiry of 1917 reported the results of the treatment of 265,158 patients by 1,268,946 injections. Among this number seventeen deaths were reported as being directly due to the drug. Of these twelve were due to *encephalitis hæmorrhagica* and four were looked on as being due to acute arsenical poisoning. Among twenty-six deaths in which "Salvarsan" could not be excluded as the cause, five were due to acute dermatitis and three to acute yellow atrophy of the liver.

Analysis of these figures shows that the greatest number of deaths occurred in the British military hospitals, even if allowance be made for the non-inclusion of dermatitis and acute yellow atrophy of the liver in the German reports. It is pointed out that a larger number of injections were given to each patient in the military institutions and that treatment was more intensive.

### Ill-Effects Other Than Those Connected with the Liver.

#### *The Immediate Ill-Effects.*

The mildest and most common symptom-complex consists in flushing, dilated pupils, slightly increased pulse rate and some dimness of vision. Urticaria may occur. Pallor and a feeble pulse are generally the precursors to vomiting. In another type of reaction rigor, fever and headache occur a few hours after the injection. These symptoms are usually mild and, although sometimes alarming, are seldom fatal. They may occur after administration of any of the arseno-benzol compounds, but are sometimes provoked by improperly prepared solutions or faulty samples of drug. In the discussion of the pathology of these immediate ill-effects, reference is made to the view of some observers that a reaction of an anaphylactic type is concerned in their production. It is pointed out that there is evidence to show that these effects may appear after the first injection of "Salvarsan." There is, moreover, held to be a relationship between the rapidity with which a sample of "Salvarsan" is precipitated from its solution and its liability to cause symptoms on injection. Reference is made to the work of Danysz, who showed that all products of arseno-benzol gave precipitates in the presence of chlorides, oxalates, carbonates, sulphates and especially alkaline phosphates. If these precipitates formed quickly, they became extremely toxic to laboratory animals. Reference is also made to the work of Widal, who held that flocculation or precipitation in the body fluids was at the root of the matter. The conclusion is reached that the terms "anaphylactic" and "anaphylactoid" should be avoided. It is held that these phenomena are of a similar nature to the experimental effects of injecting into the circulation various colloidal solutions which have the property of forming precipitates with the blood.

#### *Encephalitis Hæmorrhagica.*

The condition described as *encephalitis hæmorrhagica* has been met after the use of various arseno-benzol products. As a rule the symptoms commence suddenly within two to five days of the injection; a fatal termination

[1] "Toxic Effects Following the Employment of Arseno-Benzol Preparations" Reports of the Salvarsan Committee, Medical Research Council of the Privy Council; 1922. London: Published by His Majesty's Stationery Office; Royal 8vo., 94 pages. Price: 2s..

generally occurs within twenty-four to forty-eight hours of the onset. In a few instances the onset has occurred after the first, but, as a rule, it appears after the second injection. The clinical picture is generally characterized by intense headache followed by fever, rigors and vomiting. Epileptiform convulsions and unconsciousness are followed by coma and death. Two views have been held in regard to the pathology of this condition. According to the first view, it is really an acute cerebral syphilis of the nature of a Herxheimer reaction, intensified as a result of the injection. According to the other view, it is a direct result of the "Salvarsan," possibly in persons who are unduly susceptible to the drug. It is pointed out that the results of examination of the brain for arsenic have given such variable results that little help can be obtained by this means in the solution of the problem. An examination of the literature of acute syphilis in the pre-"Salvarsan" days does not lend support to the first theory. The Salvarsan Committee, therefore, has rejected this view. Reference is made to the work of Marschalkó and Veszprémi in their description of the *post mortem* findings in a typical instance and in their experiments on rabbits. They found hæmorrhages in various parts of the brain, sometimes small and sometimes larger. The larger ones were merely aggregations of smaller hæmorrhages. Most of the lesions were found in the *pons Varolii*. They were less abundant in the *corpus callosum*, temporal lobes and lenticular nuclei. There were no signs of either softening or inflammatory change. Microscopically numerous small hæmorrhages were seen. These consisted of red corpuscles and very few leucocytes. There was no sign of breaking down of brain cells or of cerebral softening. Apart from hæmorrhages some capillaries were seen to be filled with hyaline thrombi. Some capillaries contained aggregations of red cells and in others a typical parietal thrombosis was seen. The thrombus consisted partly of blood platelets, partly of platelets combined with fibrin and partly of leucocytes. No thrombi were found in lung, spleen, liver, kidney or stomach. Marschalkó and Veszprémi produced identical appearances in the brains of rabbits injected with 0.11 to 0.12 gramme of "Salvarsan" per kilogram of body weight. Reference is also made to the findings of Jersild, who described multiple small hæmorrhages in other organs in addition to the typical changes in the brain in a patient who had died of *encephalitis hæmorrhagica*. Stühmer found œdema of the brain in three patients at *post mortem* examination. He considered that œdema always preceded hæmorrhage.

### *Rashes and Exfoliative Dermatitis.*

The severity of skin rashes after the injection of arseno-benzol compounds varies between wide limits. In Great Britain and America where *encephalitis hæmorrhagica* is rare, exfoliative dermatitis is perhaps the commonest cause of death following the use of these arsenical preparations. Many observers have stated that skin reactions occur with the greatest frequency after the third injection. Different phases of the skin reaction may succeed one another with great rapidity or the course may be much slower and all varieties of reaction may be present on different areas of skin at the same time. The most common form of skin reaction is erythema. This may vary in intensity. It may be scarlatiniform, morbilliform, erysipelatoid or more rarely of a blotchy type. Infiltration of the skin may occur and a papular type of rash appear. This may be a small, discrete, papular rash, but more usually an urticarial type is seen. The true wheal-like character of urticaria is rarely present. Itching is very variable and may or may not be prominent. With the development of a generalized exfoliation, fever, malaise and headache occur. In severe exfoliative dermatitis emaciation may become extreme. Of the complications, glandular enlargements with abscess formation in the groins and axillæ are the most common. Broncho-pneumonia and œdema may supervene and bring about a fatal result. More rarely the eruption may take on a pemphigoid character and one instance has been reported of the occurrence of Raynaud's syndrome with gangrene.

In regard to the pathology of skin reactions it is generally held that they are the direct effects of the arsenical drugs employed. They are manifestations of arsenical poisoning appearing unduly early in persons unusually susceptible to the drug. Reference is made to the description by Moore and Keidel of peculiar changes in the bone marrow and circulating leucocytes in exfoliative dermatitis. These observers endeavoured to correlate these changes with those of fatal aplastic anæmia, a rare occurrence after the administration of drugs of the arseno-benzol type. They found a leucopenia with a decrease in the neutrophile polymorpho-nuclear cells, a definite eosinophilia and an increase in the mononuclear cells.

It is pointed out that the greatest care and judgement are necessary on the appearance of a skin reaction and the advisability of interruption or discontinuance of the arseno-benzol treatment should be considered.

### *Polyneuritis.*

Polyneuritis is a somewhat rare complication of arseno-benzol treatment. In the majority, but not in all instances, it has been preceded by a dermatitis. It is exactly similar to the polyneuritis of chronic arsenical poisoning.

### *Ulcerative Enteritis.*

Ulcerative enteritis is a rare complication. It is of the same nature as the changes in the skin in exfoliative dermatitis.

### *Damage to the Kidneys.*

No records were found by the Committee suggesting that a glomerulo-nephritis occurs as a result of arseno-benzol administration. The existence of an acute syphilitic nephritis has been proved and in the treatment of this condition injections of arseno-benzol compounds have been given with benefit. Evidence of damage to the renal epithelium has been found. Necrotic changes have been produced in the kidneys of rabbits by the injection of arseno-benzol compounds. The signs of renal damage are albuminuria, hæmaturia and sometimes suppresssion of urine. The existence of a true nephritis must be looked on as an indication for caution in using arseno-benzol compounds.

## Disorders of the Liver Following the Administration of Arseno-Benzol Preparations.

The disorders of hepatic function vary in intensity. Acute yellow atrophy of the liver appears in continental literature along with *encephalitis hæmorrhagica* as the most fatal complication of the "Salvarsan" treatment of syphilis. The Committee, however, is satisfied that dermatitis and its complications are responsible for a much larger number of fatalities than is supposed. The admittedly unsatisfactory classification has been adopted in regard to these liver conditions of (*i.*) early jaundice, (*ii.*) late jaundice and (*iii.*) acute yellow atrophy of the liver. It is pointed out that the last named condition may occur early and that benign jaundice may occur late.

### *Early Jaundice.*

Jaundice may occur within a few days or a few hours of the injection. There is no constitutional disturbance and, as a rule, the hepatic function apparently is quickly readjusted. Its ætiology is not fully understood. Recent evidence, however, suggests that even without symptoms there may be interference with liver function after every course of arseno-benzol preparations.

### *Late Jaundice.*

By the term late jaundice is understood a much more serious disorder of the liver with severe jaundice. The length of time taken for the appearance of the jaundice after the injection makes it difficult to prove that "Salvarsan" is the causative factor. The same delay is often seen in trinitrotoluene poisoning. Late jaundice is not fatal unless complicated by the development of acute yellow atrophy of the liver.

### *Acute Yellow Atrophy.*

Acute yellow atrophy of the liver generally appears to be superimposed on the condition described as late jaundice. The clinical features and *post mortem* appearances

differ in no material respect from those associated with acute yellow atrophy occurring apart from the administration of "Salvarsan." Clinically it is found that from a condition of afebrile jaundice there developes rapidly a state of acute hepatic insufficiency. Fever, rigors, vomiting, delirium and other nervous symptoms appear. These are followed by coma and death. Instances of acute yellow atrophy, as a rule, occur singly, but reference is made in the report to several groups of patients who were affected. Silbergleit and Föckler reported one outbreak and another outbreak of acute yellow atrophy occurred in Cherryhinton Hospital. A condition somewhat similar to acute yellow atrophy was reported during the war in connexion with munition workers who handled tetrachlorethane and trinitrotoluene. The same delayed action of the poison was often a definite feature. Apart from this, it would appear from the statistics collected by the Salvarsan Committee that jaundice and acute yellow atrophy have been increasing during a period of time corresponding to the "Salvarsan" era and more particularly during a period corresponding to the late war. A good account is given of the occurrence of acute yellow atrophy before the days of "Salvarsan." It is impossible to go into this aspect in any detail. It must suffice to point out that Legg collected records of one hundred instances of acute yellow atrophy and that eight of these occurred in patients who were known definitely to be syphilitic. The Committee has reviewed the opinions of many observers in regard to the occurrence of jaundice and acute yellow atrophy. Citron could not hold "Salvarsan" in any way responsible on account of the frequency of acute yellow atrophy before the days of "Salvarsan." Fabry held that the condition was due to the fixation of arsenic to the cells of the liver. Milian maintained that he could cure jaundice by giving more arsenic. Umber held that the frequency of acute yellow atrophy during the war was related to conditions apart from syphilis, such, for example, as bad feeding. Pulvermacher thought that "Salvarsan" could only be an indirect or predisposing cause in the complicated ætiology of hepatic disorders. Fraenkel held that infection by micro-organisms was the essential cause. He formed this opinion as a result of finding *Bacillus coli hæmolyticus* and *Bacillus paratyphosus B* in blood and bile when examined after death. Andrewes failed to find organisms in the blood of a patient with acute yellow atrophy. Foulerton accepted arseno-benzol compounds as the cause of acute yellow atrophy. He held that the poison was accumulated in the body fat and liberated suddenly with a variation in the nutrition of the body.

Having regard to the known hepatic insufficiency in jaundice, some work on the question has been done for the Salvarsan Committee by Dr. Mackenzie Wallis. This worker has investigated the function of the liver by means of two tests, the lævulose tolerance test and the estimation of the lipase or fat-splitting ferment normally present in the blood. Wallis found that the effects of two injections of "Salvarsan" were slight and that very little effect was found after the injection of six doses. After three months, however, definite evidence of hepatic insufficiency was almost invariably found, although no clinical evidence of hepatic disorder could be discovered. Six months after injection all signs of insufficiency had gone.

### Investigation of Two Outbreaks of Illness After Administration of Arseno-Benzol Compounds.

The Committee investigated two outbreaks of illness following the administration of arseno-benzol compounds. The first outbreak occurred at Portobello, in a military hospital. In this instance nine of eleven patients who were affected, died. It was proved that death was due to malignant malaria. The infection was handed on from patient to patient by means of blood which regurgitated into the apparatus used. This portion of the apparatus had not been submitted to sterilization in the interval between the treatment of different patients.

The second outbreak occurred at Cherryhinton Hospital. In this instance thirty-seven men treated by "Salvarsan" developed jaundice. Fifteen of them died. The larger number of these patients were confined to certain huts in the hospital. This fact created considerable difficulty in explaining the infections. The conclusion was formed that the outbreak was caused by the employment of arseno-benzol compounds, but that no special variety of the drug was more to be blamed than any other. It was held that the total dosage and the compression of the usual course into a shorter period contributed to the results. The Committee recognized there were some further factors which they could not explain.

### General Conclusions.

The Salvarsan Committee has concluded that no special arseno-benzol preparation can be regarded as more likely to produce ill-effects than others. Errors in technique cannot account for more than a few serious accidents. It is considered that excessive frequency and size of the dose may have something to do with the occurrence of ill-effects. The arsenic injected in "Salvarsan" is in the form of an organic complex which is slowly decomposed in the body. The liberation of arsenic continues for some time after the administration is completed. Many of the ill-effects may be ascribed without hesitation to the arsenic itself. Though it would seem that "shock effects" and "vasomotor" phenomena might be ascribed to capillary poisoning, there is much evidence connecting them rather with physical properties of the drug. It would seem that the phenomena have nothing to do with the presence of arsenic in the molecule, but are due to a disturbance of the colloidal state of the blood. In regard to the effects on the liver there is some difficulty. There are several facts to be considered before attaching all the blame to the drug. Acute yellow atrophy occurred frequently before the use of "Salvarsan" in conjunction with syphilis. Acute yellow atrophy often occurs at the same time as outbreaks of catarrhal jaundice in patients not under treatment for syphilis. Instances of acute yellow atrophy, moreover, occur frequently in groups of patients. The evidence pointing to the responsibility of "Salvarsan" in the causation are the evidence of deleterious effects of "Salvarsan" on the liver function, the increase of atrophy since the introduction of "Salvarsan," the constant relation in time between the administration of the drug and the occurrence of symptoms and the evidence that the pushing of the drug has been associated with an increased liability to jaundice and acute yellow atrophy. Additional evidence on this side is the fact that Hooper, Kolls and Wright have noted extensive necrosis of the centres of liver lobules as a result of the injection of "Salvarsan" in dogs.

The Committee concludes finally that many of the ill-effects are due to the arsenical content of "Salvarsan"; that others are due to its peculiar solubilities and the physical properties of its solutions and that the effects on the liver are due to the chemical nature of the whole compound, with the possibility that this type of action is dependent on the presence of other adjuvant circumstances of a nature which is at present unknown.

## Post-Graduate Work.

### SPECIAL LECTURES IN MELBOURNE.

The announcement was made in The Medical Journal of Australia of February 3, 1923 (page 138), that the Melbourne Permanent Committee for Post-Graduate Work had made arrangements with Dr. J. F. Wilkinson for a series of eight lectures on the disorders of the stomach, duodenum and pancreas.

The Committee have now completed their arrangements. The first lecture will be delivered on Tuesday, June 12, 1923, at 8.30 p.m.. The remaining seven lectures will be given on consecutive Tuesdays at the same time. All the lectures will be delivered at the Walter and Eliza Hall Institute for Research in Pathology and Medicine, Melbourne Hospital. The fee for the course will be two guineas. The title of each lecture will be published at a later date.

The Committee propose to issue to members of the class a short synopsis of the lectures at the termination of the course.

# British Medical Association News.

## SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Lister Hall, Hindmarsh Square, Adelaide, on February 22, 1923, DR. W. A. VERCO, the Honorary Treasurer, in the absence of the President, in the chair.

### A Doctor's Account of 1821.

DR. C. DUGUID, D.S.O., exhibited a doctor's account, dated 1821. He had received this account for one of his patients. The items for which charges were made, were given in detail. The commonest item was a purge, for which a charge of one shilling was made. Dr. Duguid's patient's grandfather's birth was also on the account; the fee charged was fifteen shillings. This was the largest charge made. Sums of sixpence and one shilling were the charges for visits and dressings.

### Papilloma of the Kidney.

DR. BRONTE SMEATON showed a very small papilloma of a kidney. The growth was very like the ordinary papilloma of the bladder. He said that these growths were liable to become carcinomatous.

### Hypernephroma.

DR. SMEATON also showed a hypernephroma. It had been shown in recent times that hypernephromata were in reality carcinomata.

### Renal Skiagrams.

DR. H. CAREW NOTT exhibited skiagrams of various renal conditions. He stated that a skiagram in which the transverse processes of the vertebræ and the edge of the ileopsoas muscle were not discernible, was of little diagnostic value. It was essential to obtain the renal outlines in whole or in part.

Skiagrams were shown to demonstrate the necessity of a thorough preparation of the patient before X-ray examination of the urinary tract. One film was exhibited to show the distinct renal outlines when the intestinal tract was empty, while another illustrated how fæcal and gaseous bowel contents could completely obscure the renal outlines in an otherwise good skiagram.

Dr. Nott further maintained that separate films should invariably be taken at full inspiration and expiration, so that the respiratory movement of any suspicious shadows could be studied in relation to the kidney movement. In support of this contention illustrative skiagrams of renal calculi, which maintained their relative position in the renal outline in inspiration and expiration, were contrasted with extra-renal opacities which did not.

Other skiagrams demonstrated clearly the presence of calculi in the kidney substance or in the renal pelvis. One set, in particular, showed an elongated calculus which tilted outwards as the kidney moved down, proving it to be in the ureteral orifice, whence it was subsequently extracted.

A further skiagram revealed a partially calcified kidney, deformed and lobulated on one side, and an enlarged kidney on the opposite side. Dr. Nott considered it to be probably a healed tuberculous kidney with compensatory hypertrophy of the opposite organ.

To illustrate the fact that with modern apparatus obesity was no bar to efficient radiography, a skiagram was exhibited of a very stout subject with whom it had been necessary to employ a film measuring forty-eight centimetres by thirty centimetres placed cross-ways. The renal outlines were clearly defined.

All the films had been taken with a Potter-Bucky diaphragm.

### A Typhus-Like Affection.

DR. F. S. HONE read a paper entitled "A Further Series of Cases Closely Resembling Typhus Fever" (see page 435).

DR. LIONEL B. BULL also read notes on the serological tests carried out in connexion with Dr. Hone's cases (see page 443).

DR. STEELE SCOTT said that it was a great relief to find that at last this type of case could be definitely diagnosed, thanks to the work of Dr. Hone. The general practitioner was often worried when he found himself unable to arrive at a definite diagnosis, especially in affections of the type of those deal with by Dr. Hone. He expressed his indebtedness to Dr. Hone.

DR. J. P. O'BRIEN said that he had been much impressed by the fact that his patient had not appeared to be greatly weakened, even after several weeks of acute illness.

DR. W. A. VERCO thanked Dr. Hone and Dr. Bull for their papers.

## RETIREMENT OF DR. S. W. PATTERSON.

DR. S. W. PATTERSON, the Director of the Walter and Eliza Hall Institute for Research in Pathology and Medicine, Melbourne Hospital, has tendered his resignation to the Trustees in order to take up the position of Director of the Duff House Research Hospital, North Wales. The question of Dr. Patterson's resignation was discussed at a meeting of the Council of the Victorian Branch of the British Medical Association on April 12, 1923, when the following resolution was carried without dissent:

> That on the occasion of Dr. S. W. Patterson's departure from Victoria the Council expresses its admiration of the work he has conducted at the Walter and Eliza Hall Institute and the stimulus he has provided to the whole profession for the promotion of research and the attainment of the highest standard of medical work. Also that Dr. Patterson be warmly thanked for his constant active cooperation in the scientific meetings of the Branch.

## NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

RYAN, HAROLD ALEXANDER, M.B., Ch.M., 1922 (Univ. Sydney), Bunnerong Road, Matraville.

THE undermentioned has been elected a member of the Queensland Branch of the British Medical Association:

McLEOD, ALLAN PETER, M.B., Ch.M., 1921 (Univ. Sydney), Thompson Street, Drummoyne, New South Wales (temporarily).

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

AITCHISON, RODERICK JOHN, M.B., B.S., 1922 (Univ. Melbourne), 273, Bay Street, Brighton.

HODGES, GEORGE AGINCOURT, L.R.C.P. *et* S., Edin.; L.R.F.P.S., Glas., 1913, Upper Hawthorn.

MAHON, JAMES EDMUND, M.B., B.S., 1920 (Univ. Melbourne), Ringwood.

SCANTLEBURY, GEORGE CLIFFORD, M.B. *et* Ch.B., 1915 (Univ. Melbourne), 12, Collins Street, Melbourne.

THE undermentioned has been elected a member of the Western Australian Branch of the British Medical Association:

LE SOUEF, LESLIE ERNEST, M.B., B.S., 1922 (Univ. Melbourne), Perth.

## NOTICES.

THE COUNCIL OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION has arranged the following provisional programme of the Branch meetings. The Scientific Committee reserves to itself the right to modify the arrangements, but it is hoped that no changes will be necessary.

**May 2, 1923.**

*At the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne Hospital, at 8.15 p.m.*.. DR. KEITH FAIRLEY: "Leucocytes in Health and Disease."

# Hospitals.

## CRICKET MATCH.

After an interval in some instances of twelve months, cricketing flannels were dragged out again into the light of day and the whole cricketing armamentarium was resurrected on Wednesday, March 21, 1923, on the occasion of the annual struggle between the medical staffs of the Sydney and Royal Prince Alfred Hospitals. With becoming dignity and an evident sense of their responsibility the opposing forces were marshalled after a somewhat lengthy latent period. They looked a stable and promising lot. It seems highly probable that, unless the aforementioned flannels see the light of day oftener than once a year, it will be necessary in some instances to have recourse to the compensatory gusset (we understand that that is the correct term; surgeons would probably call it a plastic graft) to provide the necessary accommodation for the wearers. The number of onlookers was small; the threatening weather may have had something to do with this. A good game was played and Royal Prince Alfred won in creditable fashion. It is rumoured that the Sydney Hospital staff has already commenced practice in the hope of turning the tables. The visitors were entertained at tea and spent an enjoyable afternoon.

The scores were as follows:

### Royal Prince Alfred Hospital.

| | Runs. |
|---|---|
| E. M. Fisher, c. W. A. Edwards, b. J. G. Edwards | 5 |
| E. H. Molesworth, c. G. C. Willcocks, b. A. J. Aspinall | 22 |
| A. H. Tebbutt, b. J. G. Edwards | 7 |
| H. R. Sear, c. R. Francis, b. J. G. Edwards | 7 |
| R. G. Waddy, c. J. M. Flattery, b. G. C. Willcocks | 18 |
| F. A. Maguire, retired | 25 |
| St. J. W. Dansey, not out | 12 |
| H. R. G. Poate, b. G. C. Willcocks | 15 |
| L. G. Teece, not out | 5 |
| Sundries | 15 |
| Total (Innings Declared Closed) | 126 |

Bowling Analysis: G. C. Willcocks, 2 for 27; J. G. Edwards, 3 for 51; A. J. Aspinall, 1 for 25; W. K. Inglis, 0 for 23.

### Sydney Hospital.

| | Runs. |
|---|---|
| J. G. Edwards, c. E. M. Fisher, b. E. H. Molesworth | 12 |
| G. C. Willcocks, c. E. H. Molesworth, b. E. M. Fisher | 22 |
| W. K. Inglis, b. E. H. Molesworth | 4 |
| W. A. Edwards, retired | 26 |
| A. J. Aspinall, b. E. M. Fisher | 5 |
| R. Francis, b. E. M. Fisher | 6 |
| J. M. Flattery, c. E. H. Molesworth, b. E. M. Fisher | 4 |
| C. Cohen, b. E. M. Fisher | 0 |
| A. J. Cunningham, b. E. M. Fisher | 0 |
| R. I. Furber, c. A. H. Tebbutt, b. E. H. Molesworth | 0 |
| G. Bell, not out | 9 |
| Sundries | 22 |
| Total | 109 |

Bowling Analysis: E. M. Fisher, 6 for 36; E. H. Molesworth, 3 for 73.

# Correspondence.

## SARCOMA OF THE MESENTERY.

Sir: In your issue of to-morrow's date is an article by Dr. Bell describing the successful removal of a sarcoma of the mesentery. It is quite typical of the general apathy towards deep X-radiation that this agency is not even suggested as a therapeutic measure. It may be that it is available or it may be that so much success has been already obtained without it that it is regarded as superfluous. Nevertheless, the tendency for sarcomata to recur and the extremely few cures of this condition are very well known indeed, yet it does not seem to be at all appreciated that an agency capable of very great amelioration and perhaps cure in certain apparently hopeless and advanced cases is surely at least as effective in early stages of the same condition and yet the bulk of malignant cases which the radiologist is expected to relieve are of the former type, whilst the more amenable cases are allowed to drift to an advanced stage without any irradiation.

That sarcomata sometimes respond to irradiation in a remarkable manner is shown in my own paper published in your journal of February 4 last. More recently, however, I have treated an extensive sarcoma of the pharynx with secondary involvement of the glands of the neck which apparently nearly disappeared in the incredibly short period of two days!

I have just received a report concerning a particularly hopeless-looking case of extensive parotid growth with numerous secondaries where I had given a very gloomy prognosis. The report is dated seven days after treatment and reads: "The parotid swelling and the glands in the neck have nearly disappeared. If I had not seen the tumour before, I would not have believed it was the same growth."

That some intra-abdominal tumours likewise do surprisingly well is also my experience, as the following examples will show:

Mrs. T., aged fifty-four years (Dr. Julian Smith), owing to jaundice *et cetera* had a laparotomy performed on September 27, 1921, but as nothing was discovered to account for her symptoms, nothing further was done at the time. Owing to a recurrence of her jaundice, she was again operated upon on July 25, 1922, when most extensive malignant disease was found about the region of her gall bladder, so that no further procedure was done except to close up the wound. She was then referred for X-ray treatment and was accordingly irradiated on August 29 and 31, 1922. Since then she had gained condition considerably, having gained at least half a stone in weight, sleeping well, with good appetite *et cetera*. On February 29, 1923, Dr. Smith reports: "I think the lesions have been held in check, but a firm ridge is to be felt transversely below ribs. Condition was a papillary growth." Weight eight stone on September 27, 1922, eight stone, seven pounds on March 6, 1923.

M.J.R., aged fifty-six years, was operated upon by Dr. Alan Newton on November 23, 1921, for intestinal obstruction, when, owing to a diagnosis of carcinoma of the splenic flexure, the transverse and pelvic colons were anastomosed. He was then transferred to the Austin Hospital for Incurables. Owing to his extremely poor condition, I hesitated to irradiate him; nevertheless, a full dose was administered on September 15, 1922, that is, nearly ten months after operation. His improvement thenceforth was very marked, for he had put on at least a stone in weight and his energy had so much improved that he kept himself occupied with gardening. Unfortunately, following an attack of pneumonia, he died on January 26, 1923. At autopsy no gross evidence of the tumour could be found, Dr. Bull reports: "There is no naked-eye evidence of malignant disease. There is subacute inflammation with effusion over the surface of the mucous membrane and the thick vessels in the specimens are rather suggestive of syphilis."

The patient had given a history of syphilis fifteen years previously. The Wassermann reaction was doubtful.

S.B., aged sixty-seven years, had had three previous operations for intestinal obstruction and was admitted to the Austin Hospital with several fæcal fistulæ over the right side of his abdomen, whence all the fæces discharged.

At the time of irradiation on October 15, 1922, he was in such great pain, owing to obstruction, that this had to be relieved by a hypodermic injection of morphine and a visiting surgeon prophesied that he would need surgical relief within twenty-four hours. Nevertheless, very soon after fæces poured forth freely from his fistulæ and he was relieved of his pain. Although at first confined to bed, he is now perfectly active, has gained at least a stone in weight and, except for the presence of the fistulæ, is practically well over five months later.

By X-ray treatment I refer only to irradiation of sufficient intensity and penetration, as anything short of this is quite useless as a therapeutic measure. High voltages are absolutely essential for efficient penetration.

I believe I have written enough about the subject of deep X-ray therapy to warrant the assumption that too little attention is paid, particularly by the teaching bodies, to it. Even the Surgical Section of the Inter-State Congress[1] cannot even find room for a discussion of this most recent advance! Yours, etc., H. Flecker.

4, Collins Street, Melbourne,
April 6, 1923.

## THE USE OF THE TOOTH BRUSH.

Sir: I read with interest and approval the letter by Dr. A. J. J. Triado on the above subject. Next day I came across an article on the care of the teeth in the women's page of a widely circulated journal. To have beautiful teeth and healthy gums it is necessary to do this: "Use a brush with good stiff bristles! . . . Scrub all your gums, starting at the very top! . . . Scrub the roof of your mouth and also your tongue! . . . If your gums bleed a little, do not be annoyed at this! . . . If you scrubbed your gums and never touched the surface of the teeth themselves, this would be better than the other way round!" According to Dr. Triado, this might be written by an unscrupulous and mercenary "pyorrhœa specialist." Yours, etc., Richard Arthur.

211, Macquarie Street, Sydney,
April 9, 1923.

[1] Dr. Flecker probably refers to the forthcoming Australasian Medical Congress (British Medical Association).

# Congress Notes.

## AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

### Entertainments.

The Executive Committee of the Australasian Medical Congress (British Medical Association) announces that arrangements for the entertainment of members are well advanced. A visit will be planned to the Zoological Gardens, where the Chief Caretaker will deliver short lectures on the various animals. Similar visits will be made to the Botanical Gardens and other places of like interest.

Arrangements are being made for visits to woollen mills, such as Foy & Gibson's, the Lincoln Knitting Company and the Yarra Falls Spinning Company. In addition, members will be able to inspect McKay's Sunshine Harvester Works, the Newport Workshops, the Pelaco Manufacturing Company's Works, the Metropolitan Fire Brigade Stations, some of the reservoirs connected with the Melbourne Water Supply, the Werribee Sewerage Farm and other important industrial undertakings.

### Memorial to Fallen Medical Officers.

The Victorian Branch of the British Medical Association has in preparation a memorial to members of the Branch who gave their lives in the Great War. This memorial will be unveiled on Monday, November 12, 1923, after the ceremony of the opening of the new Anatomy Buildings of the University of Melbourne.

### Accommodation.

Members intending to join the Congress are again reminded that there is a limited amount of good accommodation in Melbourne. They are urgently recommended to apply without further loss of time to the managers of hotels for rooms. The subjoined list is reprinted for the convenience of those who have not yet secured accommodation. We understand that nearly all the rooms at Scott's Hotel have been booked.

| House. | Address. | Persons to Accommodate. | Rooms. | | Tariff. | |
|---|---|---|---|---|---|---|
| | | | Double | Single. | Per Day. | Per Wk. |
| Scott's | Collins Street West, Melbourne | 26 | 6 | 14 | 23s. | |
| Windsor (formerly Grand) | Spring Street, Melbourne | 200 | — | — | 20s. | |
| Carlyon's | Spencer Street, Melbourne | 30 | — | — | Single Room, 5s.; Double Rooms, 10s.; Meals as taken | |
| Union Club | Collins Street West, Melbourne | 6 | — | — | 20s. | |
| New Treasury | Spring Street, Melbourne | 20 | — | — | 16s. | £4 4s. |
| Port Phillip | Flinders Street, Melbourne | 30 | — | — | 15s. | |
| Victoria Coffee Palace | Collins Street East, Melbourne | 200 | — | — | Single Rooms, 3s. 6d. and 4s. 6d.; Double Rooms, 6s., and 10s.; Meals as taken | |
| Federal Palace | Collins Street West, Melbourne | 200 | — | — | 11s. 6d. to 20s. | |
| Cathedral Hotel | Swanston Street, Melbourne | 12 | — | — | 10s. 6d. | £3 3s. |
| Esplanade (Carlyon's) | Esplanade, St. Kilda | 6 | — | — | 20s. | |
| George | Fitzroy Street, St. Kilda | 40 | 20 | — | 15s. | |
| Ritz | Fitzroy Street, St. Kilda | 30 | — | — | 20s. | |
| Prince of Wales | Fitzroy Street, St. Kilda | 30 | 15 | — | 13s. 6d. | |
| Beaconsfield | Beaconsfield Parade, St. Kilda | 25 | — | — | 15s. | |
| Inverleigh | 12, Acland Street, St. Kilda | 11 | 4 | 3 | | |
| Victoria Hotel | South Melbourne | 30 | — | — | | £5 5s. to £6 6s. |
| Bridge Hotel | Mordialloc (15 miles by electric train) | 14 | 4 | 6 | 12s. | £3 3s. |
| Hampton Hotel | Hampton (10 miles by electric train) | 12 | — | — | 15s. | £3 3s. |
| Royal Terminus (Kemp) | Brighton Beach (9 miles) | 10–12 | — | — | | £5 5s. |
| Beaumaris Hotel | Beaumaris (11 miles by motor to rail in November) | 20 | — | — | 15s. | £4 4s. |
| Mentone Hotel | Mentone (13 miles by electric train) | 20–50 | — | — | 15s. | £4 4s. |

## Obituary.

CHARLES TIMON LANE.

It is with regret that we announce the death at Brisbane on April 6, 1923, of Dr. Charles Timon Lane, who graduated at the University of Melbourne in 1885.

## Books Received.

A SIMPLE TREATMENT OF TUBERCULOSIS, by Owen F. Paget, M.D., with an Introduction by J. George Adami, M.D., F.R.S., and Prefatory Remarks by W. P. Birmingham, B.A., M.D.; 1923. London: Constable & Company, Limited; Sydney: Angus & Robertson, Limited; Crown 8vo.. pp. xvii. + 80. Price: 6s. 6d. net.

CLINICAL LABORATORY METHODS, by Russell Landram Haden, M.A., M.D.; 1923. St. Louis: C. V. Mosby Company; Demy 8vo., pp. 294, with five coloured plates and 69 illustrations in the text. Price: $3.75.

IMPOTENCY, STERILITY AND ARTIFICIAL IMPREGNATION, by Frank P. Davis, Ph.B., M.D.; Second Edition, Revised and Enlarged; 1923. St. Louis: C. V. Mosby Company; Post 8vo., pp. 168. Price: $2.25.

## Medical Appointments.

Dr. M. D. Silberberg (B.M.A.) has been appointed Lecturer in Medicine to the Australian College of Dentistry, Melbourne.

* * * *

Dr. H. G. D. Breidahl (B.M.A.) has been appointed under the *Workers' Compensation Act, 1915*, Certifying Medical Officer at Stawell, Victoria.

* * * *

Dr. Edward Field (B.M.A.) has been appointed Public Vaccinator at Glenhuntly, Victoria.

* * * *

Dr. Ellen Maude Wood (B.M.A.) and Dr. B. R. Overend (B.M.A.) have been appointed Government Medical Officers at Binalong and Trundle respectively in New South Wales.

* * * *

Dr. R. D. Fisher (B.M.A.) has been appointed Public Vaccinator at Smythesdale, Victoria.

* * * *

The undermentioned have been appointed to act as Commonwealth Medical Inspectors of Shipping and Medical Inspectors of Seamen: Dr. L. P. Brent (B.M.A.), Dr. A. J. McShane (the latter at the Port of Brisbane only).

* * * *

The appointment has been approved of Dr. J. F. M. Payne as Quarantine Officer at Devonport, Tasmania.

* * * *

Dr. G. A. Lendon (B.M.A.) has been appointed Honorary Assistant Physician at the Adelaide Hospital, *vice* Dr. D. R. W. Cowan (B.M.A.), on leave.

* * * *

Dr. A. M. Morgan (B.M.A.) has been appointed an Examiner under the provisions of the *Opticians Acts, 1920 and 1922*, of South Australia.

* * * *

Dr. John Coffey (B.M.A.) has been appointed Health Officer, Department of Public Health, Queensland.

* * * *

Dr. H. W. Tilling (B.M.A.) has been appointed a Health Officer for the purposes of *The Health Acts, 1900 to 1922*, of Queensland.

* * * *

Dr. Sydney Crawcour (B.M.A.) has been appointed Certifying Medical Practitioner in the City of Northcote for the purposes of the *Factories and Shops Acts* of Victoria.

## Medical Appointments Vacant, etc..

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

Maffrickville District Hospital: Resident Medical Officer.
Newcastle Hospital, New South Wales: Pathologist.

## Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

| BRANCH. | APPOINTMENTS. |
|---|---|
| New South Wales: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney | Australian Natives' Association<br>Ashfield and District Friendly Societies' Dispensary<br>Balmain United Friendly Societies' Dispensary<br>Friendly Society Lodges at Casino<br>Leichhardt and Petersham Dispensary<br>Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney<br>Marrickville United Friendly Societies' Dispensary<br>North Sydney United Friendly Societies<br>People's Prudential Benefit Society<br>Phœnix Mutual Provident Society |
| Victoria: Honorary Secretary, Medical Society Hall, East Melbourne | All Institutes or Medical Dispensaries<br>Australian Prudential Association Proprietary, Limited<br>Manchester Unity Independent Order of Oddfellows<br>Mutual National Provident Club<br>National Provident Association |
| Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane | Brisbane United Friendly Society Institute<br>Stannary Hills Hospital |
| South Australia: Honorary Secretary, 12, North Terrace, Adelaide | Contract Practice Appointments at Renmark<br>Contract Practice Appointments in South Australia |
| Western Australia: Honorary Secretary, Saint George's Terrace, Perth | All Contract Practice Appointments in Western Australia |
| New Zealand (Wellington Division): Honorary Secretary, Wellington | Friendly Society Lodges, Wellington, New Zealand |

## Diary for the Month.

Apr. 21.—Western Suburbs Medical Association, New South Wales: Annual Meeting.
Apr. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
Apr. 25.—Victorian Branch, B.M.A.: Council.
Apr. 25.—Western Medical Association (Blayney), New South Wales.
Apr. 26.—South Australian Branch, B.M.A.: Branch.
Apr. 26.—Brisbane Hospital for Sick Children: Clinical Meeting.
Apr. 27.—New South Wales Branch, B.M.A.: Branch.
Apr. 27.—Queensland Branch, B.M.A.: Council.
May 2.—Victorian Branch, B.M.A.: Branch.
May 4.—Queensland Branch, B.M.A.: Branch.
May 8.—New South Wales Branch, B.M.A.: Ethics Committee.
May 9.—Western Australian Branch, B.M.A.: Council.
May 9.—Melbourne Pædiatric Society.
May 11.—New South Wales Branch, B.M.A.: Clinical Meeting.
May 11.—Queensland Branch, B.M.A.: Council.
May 11.—South Australian Branch, B.M.A.: Council.
May 15.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

## Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and book-sellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.